“PREVALENCE OF HELICOBACTER PYLORI IN CHRONIC GASTRITIS: A CROSS SECTIONAL STUDY”

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI

With partial fulfillment of the regulations
for the award of the degree of

M.S (General Surgery)

Branch-I

Government Kilpauk Medical College

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"PREVALENCE OF HELICOBACTER PYLORI IN CHRONIC GASTRITIS: A CROSS SECTIONAL STUDY"** at Govt. Kilpauk Medical College Hospital is a bonafide work of Dr. F. Mohammed Muzaffar Baig. Submitted to The Tamilnadu Dr. M.G.R Medical University in partial fulfillment of requirements for the award of the degree of M.S. BRANCH I (GENERAL SURGERY) examination to be held in MAY, 2018.

Prof. Dr. R. VASUKI, M.S.,
Professor of General Surgery
Govt. Kilpauk Medical College,
Chennai – 600 010.

Prof. Dr. R. Kannan, M.S.,
H.O.D, Dept. of General Surgery
Govt. Kilpauk Medical College,
Chennai – 600 010.

Dr. P. VASANTHAMANI.,
MD, DGO, MNAMS, DCPSY, MBA.
DEAN
GOVERNMENT KILPAUK MEDICAL
COLLEGE & HOSPITAL, CHENNAI -10.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation titled **"PREVALENCE OF HELICOBACTER PYLORI IN CHRONIC GASTRITIS: A CROSS SECTIONAL STUDY"** in the General Surgery Department at Govt. Kilpauk Medical College Hospital is a bonafide research work done by **Dr. F. Mohammed Muzaffar Baig,** post graduate in M.S. General Surgery, Government Kilpauk Medical College & Hospital, Chennai-10 under my direct guidance and supervision in my satisfaction and in partial fulfillment of the requirements for the degree of **M.S. General Surgery**.

Prof. Dr. R. VASUKI M.S.,
Professor of General Surgery,
Govt. Kilpauk Medical College,
Chennai-10

Place:

Date:

DECLARATION

I hereby declare that this dissertation titled **"PREVALENCE OF HELICOBACTER PYLORI IN CHRONIC GASTRITIS: A CROSS SECTIONAL STUDY**" at Govt. Kilpauk Medical College Hospital is a bonafide and genuine research work carried out by me in the Department of General Surgery, Government Kilpauk Medical and Hospital, Chennai-10, under the guidance of our Chief **Prof. Dr. R. VASUKI, MS.,** Government Kilpauk Medical College and Hospital.

This dissertation is submitted to **THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI** in partial fulfillment of the University regulations for the award of M.S degree (General Surgery) Branch I, examination to be held in MAY 2018.

Place: **Dr. F. MOHAMMED MUZAFFAR BAIG**

Date:

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I would like to thank God for all that he has bestowed upon me.

I would like to thank my parents and wife for making me who I am today and for supporting me in every deed of mine

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INSTITUTIONAL ETHICS COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No.06/2016 Meeting held on 14/12/2016
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "PREVALENCE OF HELICOBACTER PYLORI IN CHRONIC GASTRITIS:A CROSS SECTIONAL STUDY" submitted by Dr.F.Mohammed Muzaffar Baig.,M.S.Post Graduate,Dept of General Surgery, Govt. Kilpauk Medical College, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

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Sources included in the report:

https://en.wikipedia.org/wiki/Gastric_ulcers

Instances where selected sources appear:

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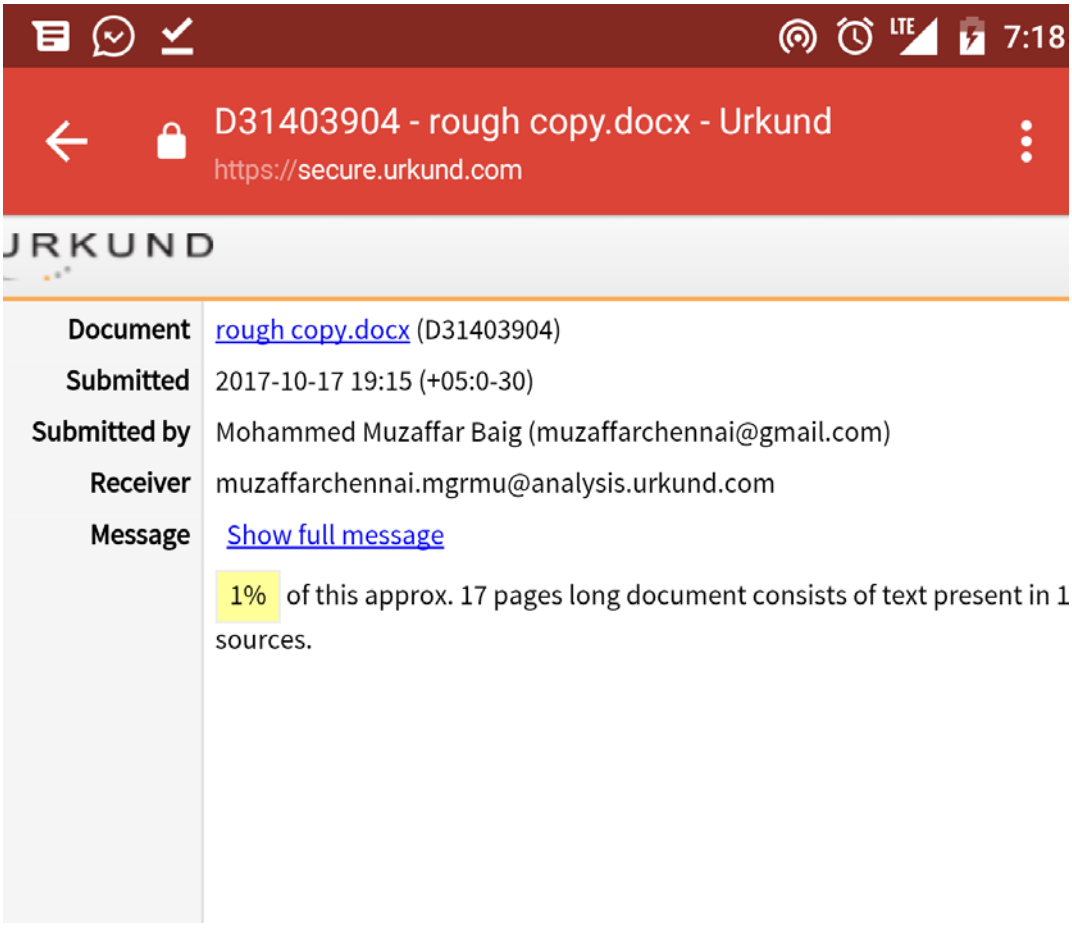


ABBREVIATIONS

AKA	Also Known As
AP	Activator Protein
BT	Bleeding Time
CDC	Centre For Disease Control
CREB	Camp Response Element Binding Protein
CT	Clotting Time
CAG	Cytotoxin Associated Gene
DNA	Deoxyribo Nucleic Acid
H. pylori	Helicobacter pylori
IL	Interleukins
MALT	Mucosa Associated Lymphoid Tissue
NF k B	Nuclear Factor Kappa B
OGD	Oesophago Gastro Duodeno
OPD	Out Patient Department
PMN	Polymorphonuclear Neutrophils

PPI	Proton Pump Inhibitors
RUT	rapid urease test
TNF	Tumor Necrosis Factor
VAC	Vacuolating Cytotoxin
VCTC	Voluntary Counselling and Testing Centre

TABLE OF CONTENTS

INTRODUCTION

Chronic gastritis and peptic ulceration are widely distributed throughout the world. *Helicobacter pylori* majorly contributes to chronic active gastritis and has serious complications like gastric adenocarcinoma and mucosa associated lymphoid tissue lymphoma. *H. pylori* is a gram negative bacilli that is usually seen in gastric pits under the mucus layer and found in close association to gastric epithelial cells. Approximately, half of the normal population across the world harbor *H. pylori*, but only 10-20% of them are symptomatic. There is a strong correlation of *H. pylori* infection with hygiene, life-style, and economy, thereby leading to an annual incidence rate of *H. pylori* infection ≈ 4 -5% in developing countries compared to that of ≤ 0.5% in developed and industrialized countries.

There are many other causative factors such as tobacco, non-steroidal anti-inflammatory drugs (NSAIDS), and gastric juice reflux (chemical gastritis) that are also associated with chronic gastritis. H. pylori along with other etiological factors contributes to chronic gastritis.

There are non-invasive tests and invasive tests for H.pylori diagnosis. Non-invasive tests include, urea breath analysis, serological Immunoglobulin G (IgG) and Immunoglobulin M (IgM) detection, saliva

and urinary antibody test, and stool antigen test. The invasive tests involve endoscopy, which include histopathological examination, culture, rapid urease test (RUT) and polymerase chain reaction. Invasive tests carry high sensitivity and specificity of >90%. The non-invasive tests such as serology is useful in areas of high prevalence, as it demarcates between previous and current infection.

The prevalence of *H. pylori* infection in different regions of the world varies. Inspite of the umpteen studies, still there is insufficient information about *H. pylori* infection prevalence in this part of Southern India. The present study was conducted to assess the prevalence of *H. pylori* infection among patients presenting with the dyspepsia and to correlate its association with chronic gastritis, in a medical college hospital in Tamilnadu, India.

AIM OF THE STUDY:

To determine the prevalence of helicobacter pylori infection in chronic gastritis.

REASON FOR CONDUCTING THE STUDY

1. Dyspepsia, accounting for a major number of cases reporting daily in surgical OPD, is becoming a common menace in general public health.

2. Chronic gastritis has become more common cause for upper abdominal pain.

3. Increased usage of antibiotics and proton pump inhibitors is being reported in all parts of India.

4. To assess the recent trend in prevalence of Helicobacter pylori in patients with chronic gastritis.

HISTORICAL BACKGROUND[1][2]

History of Ulcer Diagnosis and Treatment

A complete cure for ulcers had been an enigma for ages. As it has been observed that ulcers are caused by a bacterium, and can be cured with antibiotics. This observation has turned out to be a boon.

Early 20th Century

Ulcers were attributed to stress and dietary factors. Hospitalization, bed rest, and intake of special bland foods was advised. Later, abnormal gastric acid levels was found to be leading ulcer disease. Antacids became the standard of therapy. Despite the treatment, there was a high recurrence of ulcers.

1900

Osler and McCrae published about an acute form of gastritis with hypochlorhydria in their *Principles and Practice of Medicine* volumes.

1940

Spiral organisms observed in gastric mucosa in gastric resected specimens by Freedberg and Baron

1950

Susumu Ito of Harvard school published first detailed anatomy of gastric mucosa appearance under the electron microscope. Excellent

photograph of one of these organisms in 1967, showing a greatly enlarged, flagellated, spiral shaped *H. pylori* within a parietal cell gland,

1970

Spiral organisms were confirmed in animals by G Bizzazero

1982

Australian physicians Robin Warren and Barry Marshall were the first to identify the link between *Helicobacter pylori* (*H. pylori*) and ulcers, concluding that the bacterium causes ulcers.

1994

A National Institute of Health Consensus Development Conference concluded that there is a strong association between *H. pylori* and ulcer disease, and recommended antibiotics as the treatment of choice.

1995

Data showed that about 75 percent of ulcer patients were still treated primarily with antisecretory medications, and only 5 percent with antibiotics. Nearly 90 percent of those with ulcers blame their ulcers on stress or worry, and 60 percent pointed to diet.

1996

The Food and Drug Administration approved the first antibiotic for treatment of ulcer disease.

1997

The Centers for Disease Control and Prevention (CDC) launched a national level education campaign to inform about the link between *H. pylori* and ulcers. This campaign reinforced the news that ulcers are curable Medical researchers sequenced the *H. pylori* genome. This discovery helped scientists to design more effective drugs to fight it.

ANATOMY OF STOMACH[3][4]

The **stomach**, part of the gastrointestinal tract, is a digestive organ located between the esophagus and the duodenum.

It has a ‘J’ shape, and features a lesser and greater curvature. The anterior and posterior surfaces are smoothly rounded with a peritoneal covering.

Anatomical Position

The stomach is located in the superior aspect of the abdomen. It lies in the **epigastric** and **umbilical** regions, mostly protected by the lower portion of the rib cage.

The exact size, shape and position of the stomach can vary from person to person. For example, in thin individuals, it is not uncommon for the stomach to extend into the pelvic region.

Anatomical Structure

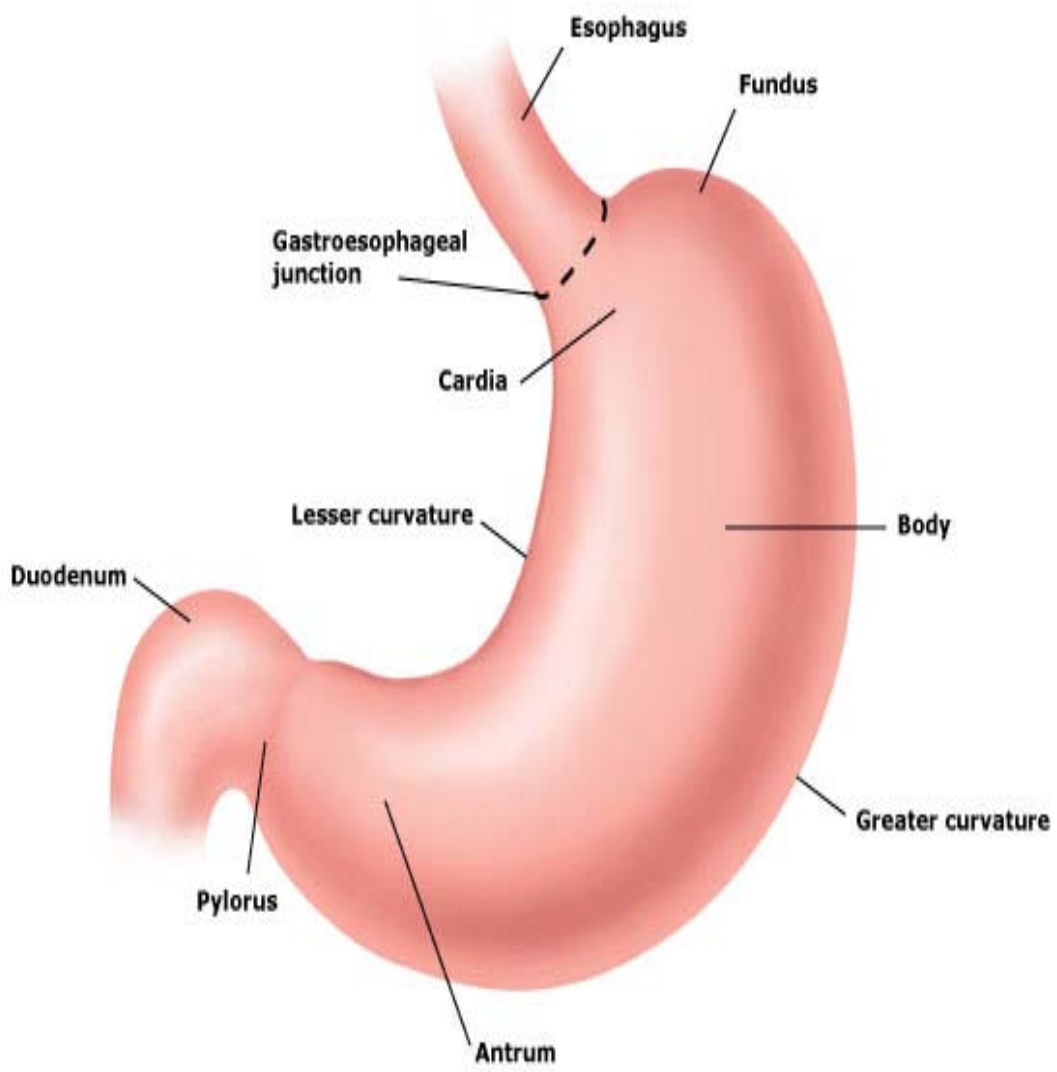
The stomach has four main regions; the cardia, fundus, body and pylorus:

Cardia – surrounds the superior opening of the stomach.

Fundus – the rounded portion superior to and left of the cardia.

Body – the large central portion inferior to the fundus.

Pylorus – connects the stomach to the duodenum**.**



Anatomy of stomach

Greater and Lesser Curvatures

The medial and lateral borders of the stomach are curved, forming the lesser and greater curvatures.

Greater curvature – forms the long, convex, lateral border of the stomach.

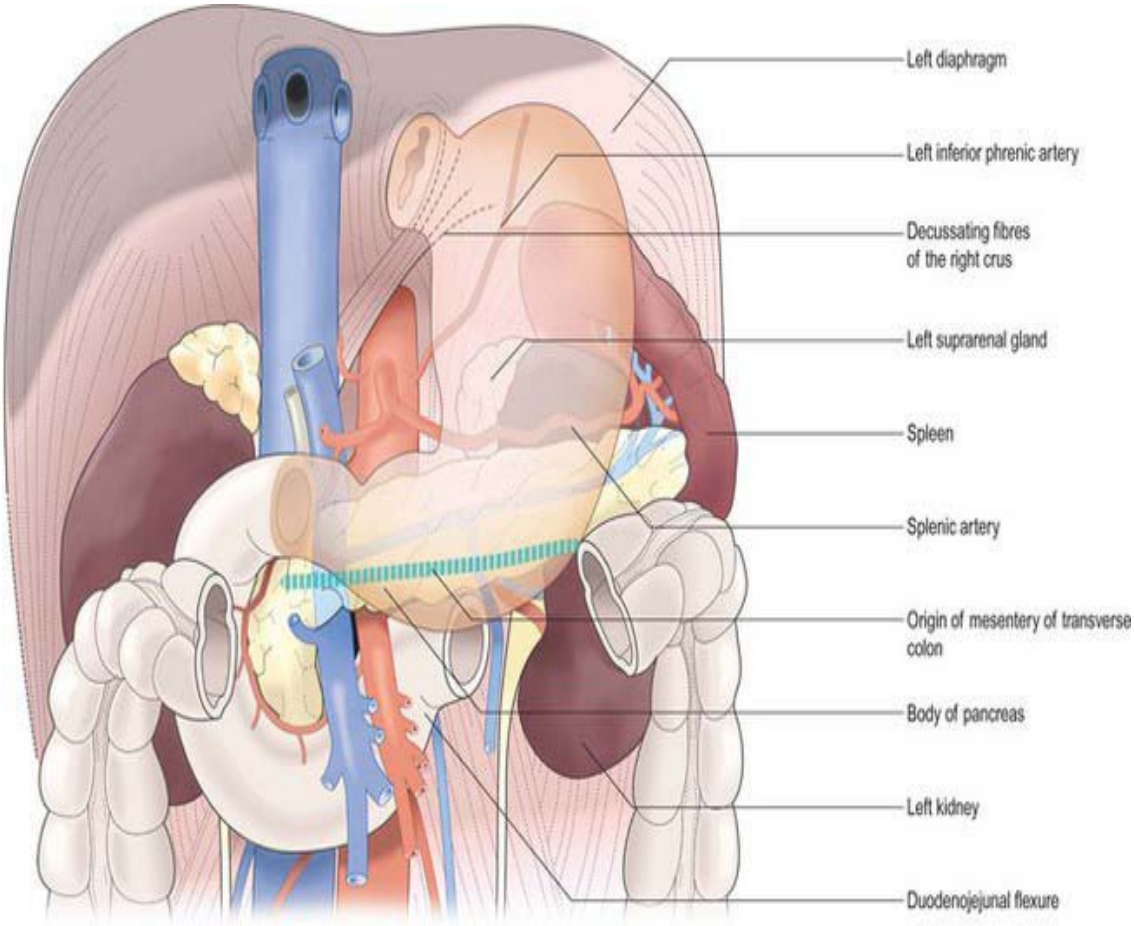
Arising at the cardiac orifice, it arches backwards and passes inferiorly to the left. It curves to the right as it continues medially to reach the **pyloric antrum**. The short gastric arteries and the right and left **gastro-omental** arteries supply branches to the greater curvature.

Lesser curvature – forms the shorter, concave, medial surface of the stomach. The most inferior part of the lesser curvature, the **angular notch,** indicates the junction of the body and pyloric region. The lesser curvature gives attachment to the **hepatogastric ligament** and is supplied by the left gastric artery and right gastric branch of the hepatic artery.

Anatomical Relations

The anatomical relations of the stomach are given in the table below:

Anatomical relation	Structures
Superior	Oesophagus, Diaphragm
Inferior	Head and neck of pancreas
Anterior	Greater omentum, abdominal wall, left lobe of liver, gall bladder
Posterior	Lesser sac, left kidney, left adrenal gland, splenic artery, common bile duct, gastroduodenal artery



Anatomical relations: Stomach

Sphincters of the Stomach

There are two sphincters of the stomach, located at each orifice. They control the passage of material entering and exiting the stomach.

Inferior Oesophageal Sphincter

The **inferior oesophageal sphincter** is located between the oesophagus and the stomach (in contrast to the superior oesophageal sphincter, located in the pharynx).

It is located to the left of the T11 vertebra. Situated immediately superior is the **oesophageal hiatus**, an opening in the diaphragm through which the oesophagus travels. Histologically, the sphincter is marked by an abrupt change from **stratified squamous** epithelium to **simple columnar**.

The inferior oesophageal sphincter is termed as **physiological** (or functional) sphincter – it does not have any specific muscle

Pyloric Sphincter

The pyloric sphincter lies between the **pylorus** and the **duodenum**. It controls of the exit of **chyme** (food and gastric acid mixture) from the stomach.

In contrast to the inferior oesophageal sphincter, this is an **anatomical sphincter**. It contains smooth muscle, which constricts to limit the discharge of stomach contents through the orifice.

Emptying of the stomach occurs intermittently when **intragastric pressure** overcomes the resistance of the pylorus. The pylorus is normally contracted so that the orifice is small and food can stay in the stomach for a suitable period. Gastric peristalsis pushes the chyme through the pyloric canal into the duodenum for further digestion.

Greater and Lesser Omenta:

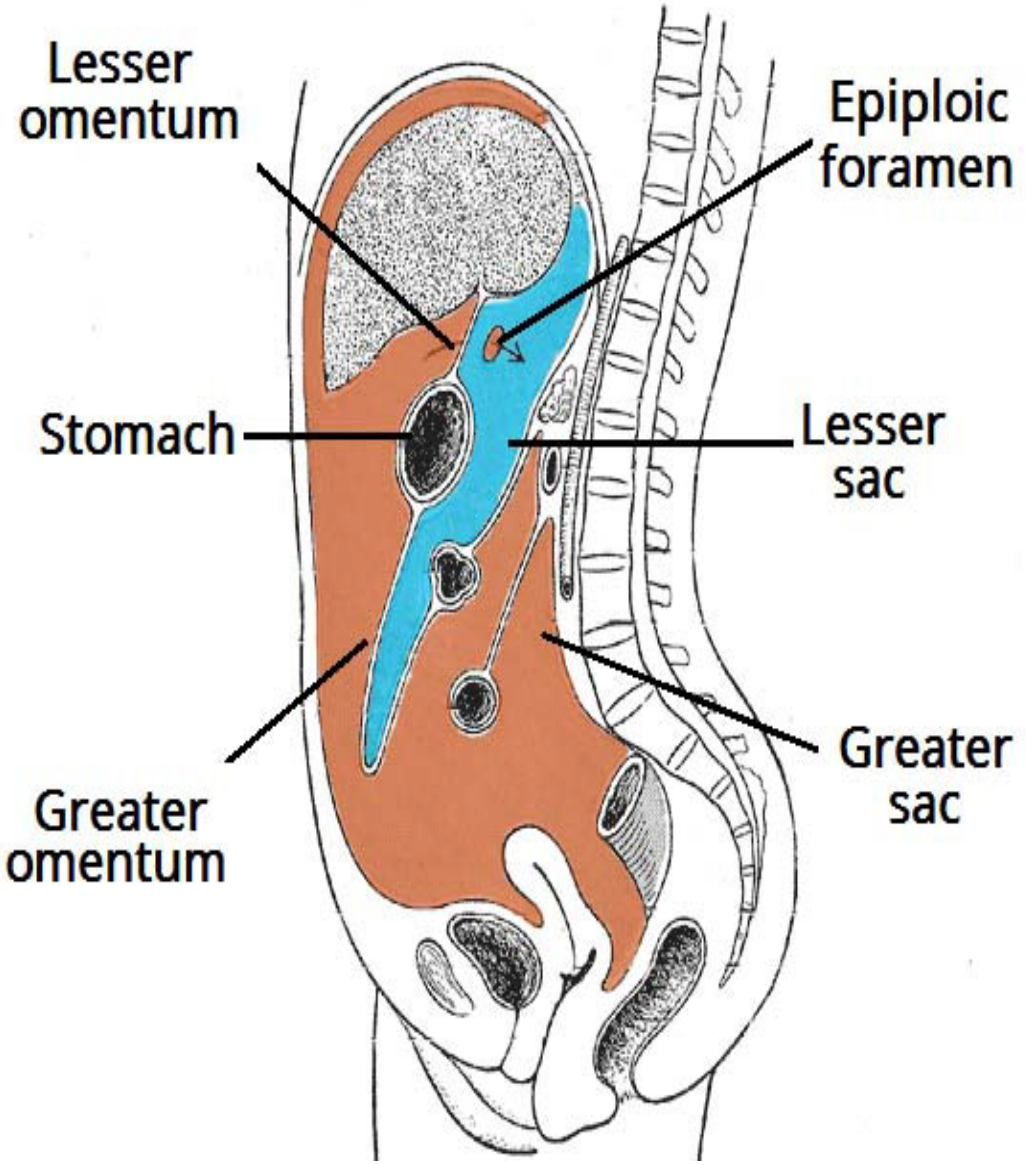
Within the abdominal cavity, the organs are covered in a double layered membrane, called the **peritoneum**. It supports the viscera, and attaches them the abdominal wall.

The **greater** and **lesser omenta** are two structures that consist of peritoneum folded over itself (two layers of peritoneum – four membrane layers). Both omenta attach to the **stomach**, and are useful anatomical landmarks:

Greater omentum – hangs down from the **greater curvature** of the stomach. It drapes over the **transverse colon** and folds back upon itself before reaching the posterior abdominal wall. It features many **lymph nodes,** which contain macrophages to help combat infections of the GI tract.

Lesser omentum – continuous with peritoneal layers of the stomach and duodenum. These two layers combine at the lesser **curvature**, and ascend to attach to the **liver**. The main function of the lesser omentum is to attach the stomach and duodenum to the liver.

Together, the greater and lesser omenta divide the abdominal cavity into two; the greater and lesser sac. The stomach lies immediately anterior to the **lesser sac.** The greater and lesser sacs communicate via the **epiploic foramen**, a hole in the lesser omentum.



Sagittal section of abdomen showing

greater and lesser, omentum and sac

Neurovascular Supply

The arterial supply to the stomach comes from the **coeliac trunk** and its branches. Anastomoses form along the lesser curvature by the right and left **gastric arteries**and along the greater curvature by the right and left **gastro-omental** arteries:

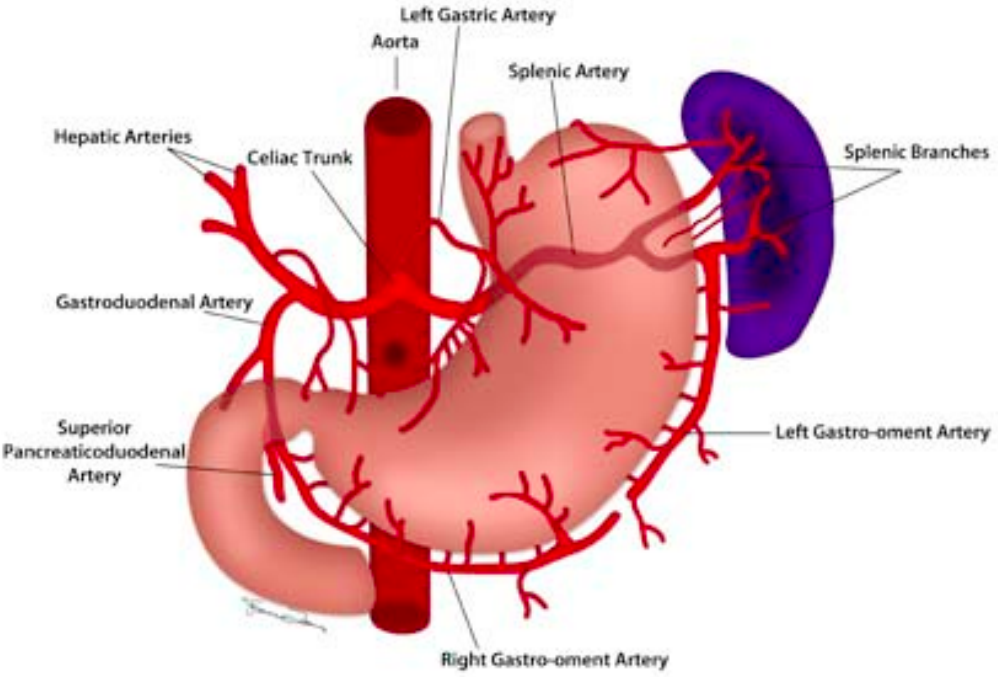
Right gastric – Branch of the common hepatic artery, which arises from the coeliac trunk.

Left gastric – Arises directly from the coeliac trunk.

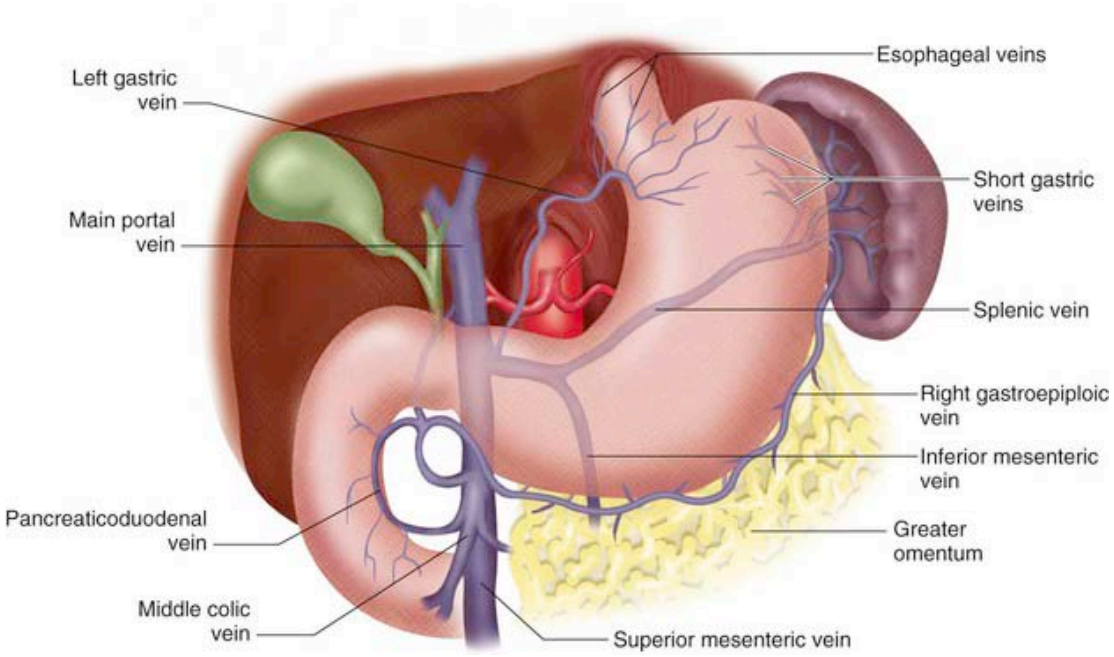
Right gastro-omental – Terminal branch of the gastroduodenal artery, which arises from the common hepatic artery.

Left gastro-omental – Branch of the splenic artery, which arises from the coeliac trunk.

The veins of the stomach run parallel to the arteries. The right and left gastric veins drain into the **hepatic portal vein**. The short gastric vein, left and right gastro-omental veins ultimately drain into the superior mesenteric vein.



Arterial supply of stomach



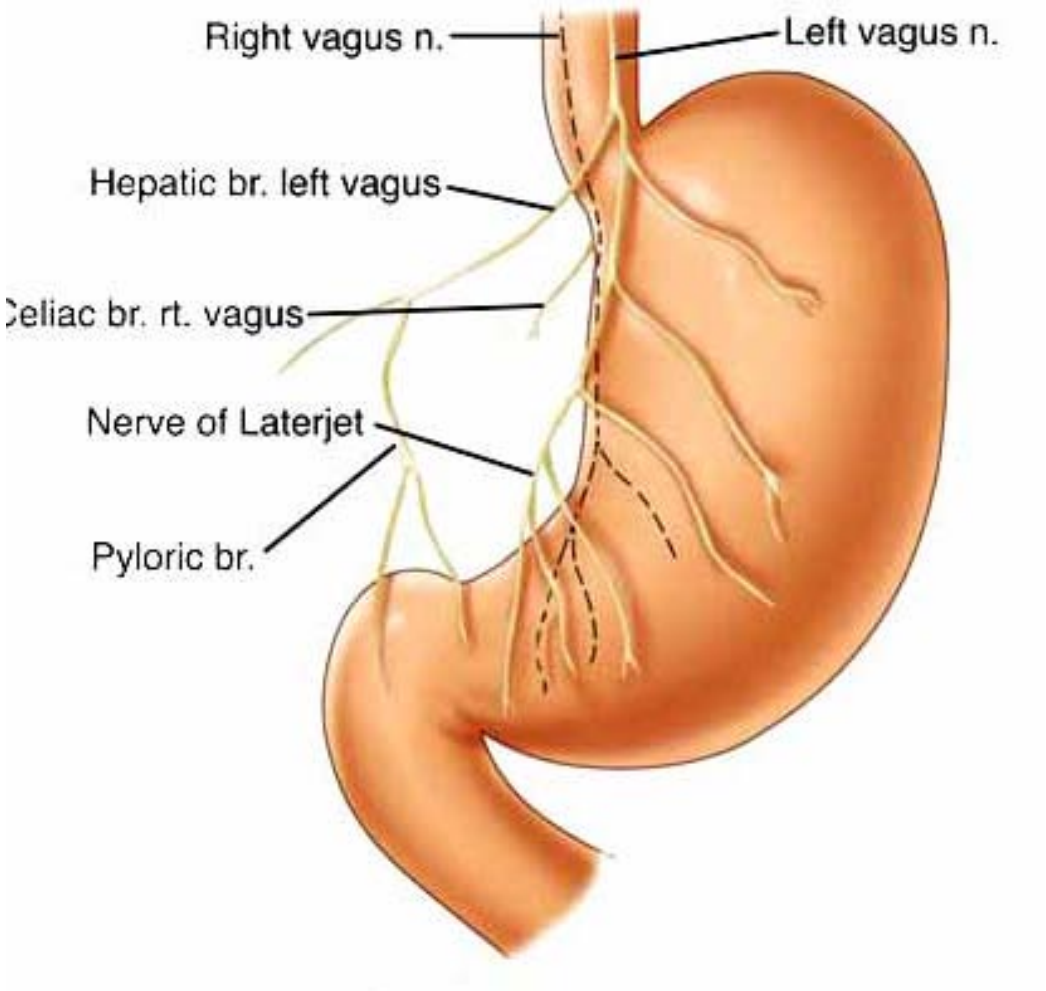
Venous drainage of stomach

Innervation

The stomach receives innervation from the autonomic nervous system:

Parasympathetic nerve supply comes from the posterior vagal trunks, derived from the vagus nerve.

Sympathetic nerve supply from the T6-T9 spinal cord segments pass to the coeliac plexus. It also carries some pain transmitting fibres.



Vagal nerve supply of stomach

Lymphatic drainage:

Lymph nodes draining the stomach are numbered and divided into 4 levels, as follows:

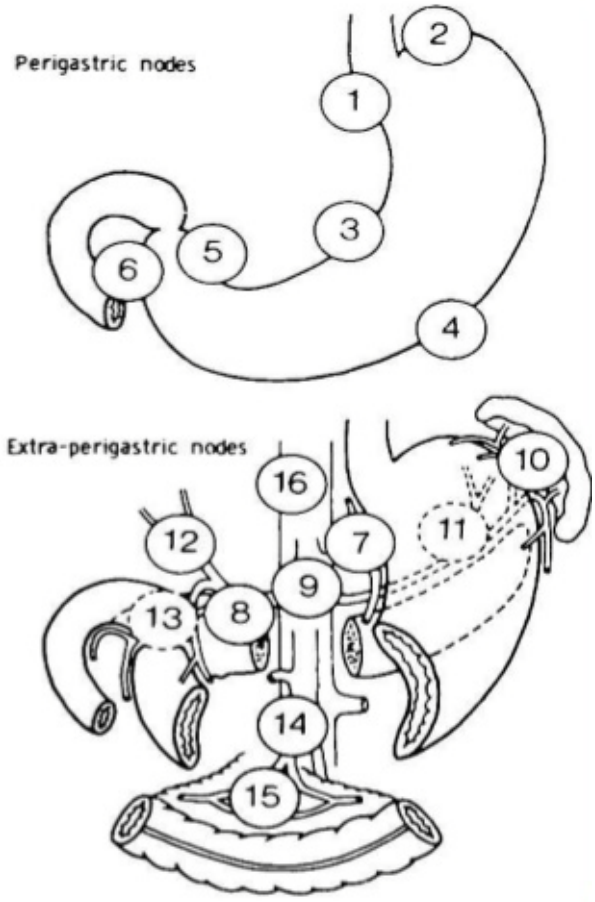
Level 1 - (perigastric lymph nodes) - Right paracardiac (1), left paracardiac (2), along lesser curvature (3) along greater curvature (4), suprapyloric (5), infrapyloric (6)

Level 2 - Along Left gastric artery (7), along Common hepatic artery (8), along celiac axis (9), at splenic hilum (10), along splenic artery (11)

Level 3 - In hepato-duodenal ligament (12), behind duodenum and pancreas head (13), at the root of small bowel mesentery (14)

Level 4 - Mesocolic (15), paraaortic (16)

LN group	
1	R cardiac
2	L cardiac
3	Lesser curvature
4	Greater curvature
5	Suprapyloric
6	Infrapyloric
7	L gastric artery
8	Common hepatic artery
9	Celiac artery
10	Splenic hilar
11	Splenic artery
12	Hepatic pedicle
13	Retroduodenal
14	Mesenteric root
15	Middle colic artery
16	Paraaortic
17	Around lower oesophagus
18	Supradiaphragmatic



Lymph node stations of stomach

Histology of stomach:

Like the other parts of the gastrointestinal tract, the stomach walls has an outer mucosa, and inner submucosa, muscularis propria, and serosa.

The gastric mucosa consists of the epithelium and the lamina propria (composed of loose connective tissue), muscularis mucosae separates lamina propria from the submucosa beneath.

The submucosa lies under the mucosa and consists of fibrous connective tissue, separating the mucosa from the muscle layer. Meissner's plexus lies in this layer. The muscularis externa lies deepto this submucosa, and is unique from other organs of the gastrointestinal tract, consisting of three layers:

The *inner oblique layer:* This layer creates the motion that churns and physically breaks down the food. It is the only layer, which is not seen in other parts of the digestive system. The antrum has thicker skin cells in its walls and performs more forceful contractions than the fundus.

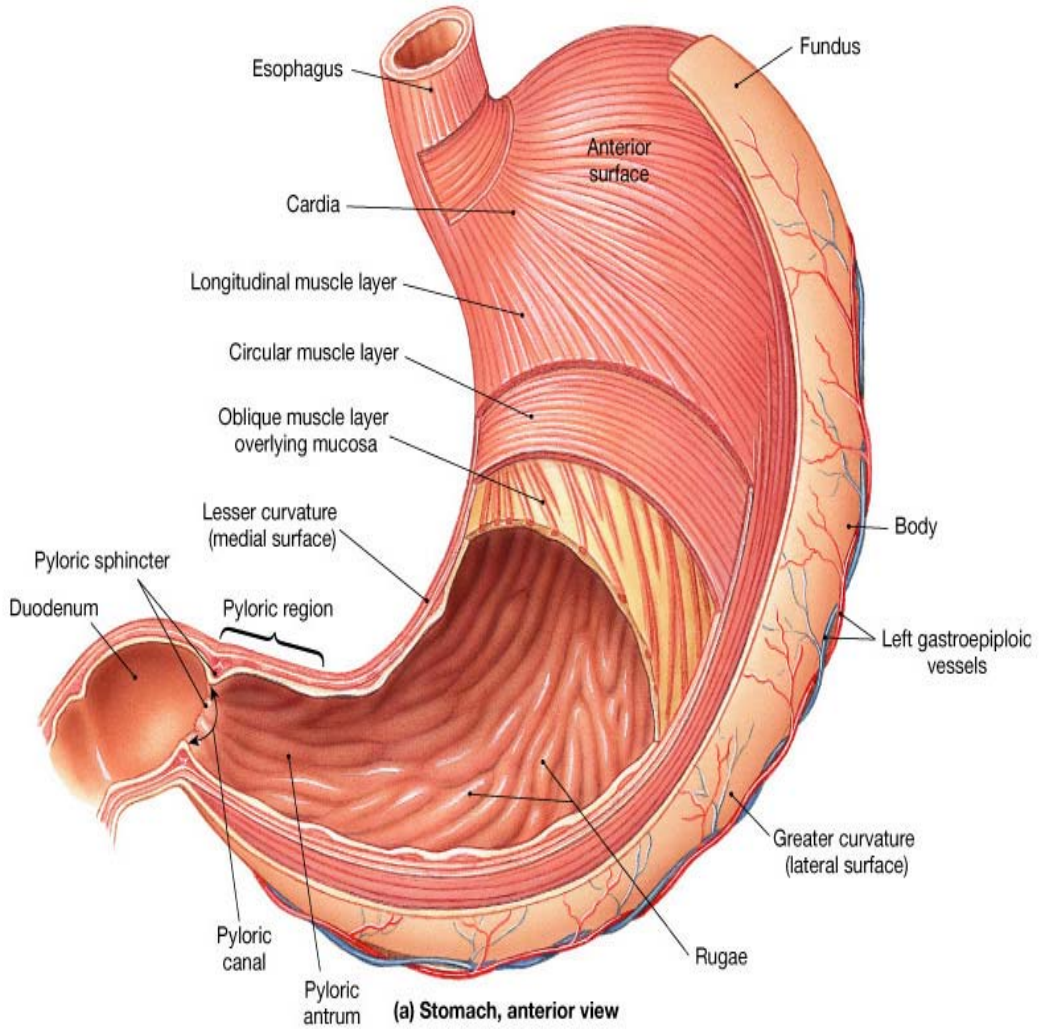
The *middle circular layer:* The pylorus is surrounded by a thick circular muscular wall which is usually tonically constricted forming a functional (if not anatomically discrete) pyloric sphincter, which controls the movement of chyme into the duodenum. This layer is perpendicular to the longitudinal axis of the stomach.

Auerbach's plexus (AKA myenteric plexus) lies between the outer longitundinal and the middle circular layer and is responsible for the innervation of both (causing peristalsis and mixing)

The *outer longitudinal layer* moves the bolus towards the pylorus of the stomach through muscular shortening.

The stomach also possesses a serosa, consisting of layers of connective tissue which is continuous with the peritoneum.

MUSCULATURE OF STOMACH



Gastric gland secretions:

GASTRIC MUCOSA	CELL TYPES	SUBSTANCE SECRETED	STIMULUS FOR RELEASE	FUNCTION OF SECRETION
	Mucous neck cell	Mucus	Tonic secretion; with irritation of mucosa	Physical barrier between lumen and epithelium
		Bicarbonate	Secreted with mucus	Buffers gastric acid to prevent damage to epithelium
	Parietal cells	Gastric acid (HCl)	Acetylcholine, gastrin, histamine	Activates pepsin; kills bacteria
		Intrinsic factor		Complexes with vitamin B_{12} to permit absorption
	Enterochromaffin-like cell	Histamine	Acetylcholine, gastrin	Stimulates gastric acid secretion
	Chief cells	Pepsin(ogen)	Acetylcholine, acid secretion	Digests proteins
		Gastric lipase		Digests fats
	D cells	Somatostatin	Acid in the stomach	Inhibits gastric acid secretion
	G cells	Gastrin	Acetylcholine, peptides, and amino acids	Stimulates gastric acid secretion

REVIEW OF LITERATURE

Studies showing no difference in gender:

Adlekha, S et al said that “we did not get a significant difference in *H. Pylori* prevalence according to gender”[5]

Tarkhashvili, Nato et al inferred that “male sex did not confer increased risk for H. pylori infection”[6]

Khan AR et al, said that “the infection affects both the genders equally, whereas gastric cancer has a male preponderance pehaps due to some additional factors”[22]

Joutei HA et al, said that “gender has no significant associataion with H. pylori infection”[19]

Fraser AG et al, said that “H. pylori infection was not significantly associated with gender, alcohol and cigarette use”[15]

Ogutu EO et al, inferred that “theres no difference in prevalence of H. pylori infection among male and female”[9]

Studies showing increased prevalence of H. pylori in 5th decade of life:

Baako BN et al, said that “the incidence of H. pylori infection peaks in 5th decade of life”[12]

Hashemi et al, said that “older age is independently associated with H. pylori infection which peaks in 5th decade of life”[14]

Fraser AG et al inferred that “the relative risk of H pylori infection significantly increased with age more in 40-50 years of age, lower socio-economic status and lower household income”[15]

Ortega et al, said that “the prevalence of H pylori steadily increases with age and attains a peak in 5th decade and falls down with further increasing age”[18]

Joutei et al, said that “the difference in prevalence between the age group 40-50 years and other age groups was statistically significant”[19]

Nguyen et al, said that “the prevalence of infection was significantly higher in those over 40 years of age than in those aged <40”[21]

Mbengue M et al, said that “incidence of H pylori infection starts at early age and extremely high at 40 to 50 years”[10]

Studies showing gastritis as most common endoscopic findings:

Adlekha et al said that “the commonest identifiable lesion at endoscopy was chronic gastritis and its association with H. pylori was 62.5%”[5]

Abiodun Christopher et al, said that “The most common abnormality at endoscopy was gastritis which was seen in 60.5% patients. 79.1% patients had endoscopically identifiable cause for their dyspepsia while the remaining 20.9% had normal endoscopic findings”[7]

Hashemi MR et al, said that “the most common endoscopic finding in a study population of 568 persons was gastritis followed by gastric ulcer and duodenal ulcer”[14]

Al akwaa AM et al, said that “The main endoscopic findings were gastritis in 42 (67.7%), hiatus hernia in 8 (13%), and gastric erosions in 8 patients (13%)[17]

Ortega JP et al, said that, “On performing endoscopy in dyspeptic patients, the common findings was gastritis in upto 65%”[18]

Alsaimary et al, said that “80% of patients undergoing UGI scopy had gastritis in anyone stage in clinically dyspeptic patients”[20]

Studies showing varied prevalence of H. Pylori in chronic gastritis and antral predilection:

Adlekha S et al, said that "The association of this lesion(chronic gastritis) with *H. pylori* infection was found to be statistically significant with 85.7%"[5]

Tarkhasvili et al, said that "*H. pylori* positivity was strongly associated with active inflammation in chronic gastritis in 79%"[6]

Abiodun Christopher et al, said that "This study showed that 63.5% of patients with endoscopic gastritis had *H. pylori* infection"[7]

Oluwasola et al, said that "The study shows that Helicobacter pylori infection is associated mainly with moderate to severe chronic gastritis in 74 % of cases examined. This study confirms the antral predilection of H. pylori infection, and the finding of an antral preponderance for chronic gastritis partly explaining the relatively more frequent occurrence of duodenal ulcers in Nigerians as compared to gastric ulcers"[8]

Ogutu EO et al, said that "All our cases of peptic ulcer disease and chronic gastritis had evidence of H. pylori infection in histological examination"[9]

Baako BN et al, said that “A hundred and thirty (130) patients were studied. 75.4% tested positive for H. pylori infection and all of them had chronic gastritis and the incidence peaks in the 5th decade”[12]

Al Akwaa AM et al, said that “Sixty two patients were referred for upper GI endoscopy as pre bariatric surgery evaluation. *Helicobacter pylori* infection was detected histologically in 53 patients (85.5%). All patients with positive HP organism in their biopsy specimens had chronic active gastritis”[17]

Ortega JP et al, said that “Frequency of H. pylori infection was 86.6% in chronic gastritis. Prevalence of H. pylori infection is very high in symptomatic and in those with gastroduodenal ulcer or erosions, while in patients with erosive esophagitis is similar to those with normal endoscopy”[18]

Joutei HA et al, said that “The prevalence of H. pylori infection was 69%. H. pylori infection was found in 92% of chronic gastritis cases. The prevalence of H. pylori was significantly higher in the antrum (73%) than in the corpus (21%) and the pylorus (6%).[19]

Alsaimary et al, said that “The results showed that 81% of the patients gave positive results in HPE for H. Pylori with chronic gastritis.

About 20% of people under the age of 40 and half of those over the age 40 have H. Pylori”[20]

Nguyen et al, said that “The prevalence of infection was significantly higher in those over 40 years of age than in those aged ≤40. Chronic gastritis was present in all *H. pylori*-infected individuals, 83.1% of whom had active gastritis, and 85.3% and 14.7% had atrophy and intestinal metaplasia, respectively”[21]

CHRONIC GASTRITIS

Chronic gastritis is chronic inflammation of gastric mucosa associated with damage to superficial and glandular epithelia. It is a pathophysiological diagnosis. Patients with symptoms of dyspepsia undergo upper gastroduodenoscopy and gastric biopsies, followed by which a diagnosis of chronic gastritis can be made.

The updated Sydney system grading for chronic gastritis defined chronic gastritis as chronic inflammation of gastric mucosa with increased lymphocytes and plasma cells in lamina propria[23]. Activity of chronic gastritis is graded with neutrophilic infiltration of lamina propria, pits or surfaces as mild, moderate and severe.

Helicobacter pylori is a major cause of chronic gastritis, peptic ulcer disease, gastric adenocarcinoma and primary gastric lymphoma. Initially coined by Marshall and Warren in 1983, *H pylori* is a spiral gram-negative rod that usually colonizes and infects the stomach. The bacteria lives beneath the mucous layer and the upper portions of the gastric foveolae. Children usually get infected and once present in the stomach, the bacteria invades the mucous layer and establishes itself in the luminal surface of the stomach causing an intense inflammatory changes in the underlying tissue. The *H pylori* causes tissue damage and

the histologic finding of both an active and a chronic gastritis observed. The presence of PMNs in the gastric mucosa is diagnostic of active chronic gastritis.

As a result of response to H pylori colonization in surface mucosa it leads to release of interleukin (IL)-8, which recruits Poly morphonucleocytes and may begin the entire inflammatory process. Gastric epithelial cells express class II molecules, which may increase the inflammatory response by presenting *H pylori* antigens, leading to the activation of numerous transcription factors, including NF-kB, AP-1 and CREB-1[24]. This in turn leads to further cytokine release and more inflammation. High levels of cytokines, particularly tumor necrosis factor-α (TNF-α) and multiple interleukins (eg, IL-1β, IL-6, IL-8, IL-10, IL-12, IL-17 and IL-18), are detected in the gastric mucosa of patients with *H pylori* gastritis.

Leukotriene B4 produced by host neutrophils also elevated ,which is cytotoxic to gastric epithelium. This inflammatory response produces functional changes in the stomach, depending on the areas of the stomach involved. When gastric corpus area is affected by inflammation, parietal cells are inhibited, causing decreased acid secretion. Persisting inflammation results in loss of parietal cells, and permanent decrease in acid secretion. H.pylori infected individuals will have an abnormal

gastrin secretion, especially following meal intake. As the infection gets cured, there will be quicker resolution of neutrophils followed by slower resolution of chronic inflammatory cells[25].

After resolution of chronic inflammatory cells, gastrin secretion following meal gets normalized. It is the difference in the virulence factors of the variety of strains of H.pylori that determine the course of each of these strains. *H pylori* strains releasing the vacuolating toxin A (vacA) have higher tendency to cause peptic ulcers than non-vacA producing strains[26]. Strains secreting CagA protein ($CagA^+$) have stronger association with gastric carcinoma and peptic ulcers. Even $CagA^-$ strains can lead to these diseases. [27][28]

H pylori chronic gastritis has following two courses[29][30]:

- Antral predominant gastritis: Inflammatory process involves antrum leading to peptic ulcers.
- Multifocal atrophic gastritis – Here the inflammatory process involves corpus and gastric antrum followed by atrophy (loss of gastric glands) and intestinal metaplasia , leading to gastric carcinoma and gastric ulcers .

50% of the world's population is infected with *H pylori.* Majority remain carriers with asymptomatic chronic gastritis. Additional risk factors possessing individuals may develop peptic ulcers, gastric mucosa–associated lymphoid tissue (MALT) lymphomas, or gastric adenocarcinomas.

An increased duodenal acid load leads to bile salts wash out, which normally inhibit the growth of *H pylori.* Progressive damage to the duodenum produces gastric foveolar metaplasia, results in *H pylori* growth and more inflammation. This cycle makes the duodenal bulb increasingly unable to neutralize acid entering from the stomach, causing changes in the bulb structure and function, producing ulcer. *H pylori* can live in areas of gastric metaplasia in the duodenum, contributing to the development of peptic ulcers. [31][32]

MALT lymphomas also develop in association with chronic gastritis secondary to *H pylori* infection. The stomach usually doesn't have organized lymphoid tissue, but after infected with *H pylori,* lymphoid tissue is universally present. Acquisition of gastric lymphoid tissue is because of persistent antigen stimulation from byproducts of chronic infection with *H pylori.* [33][34]

The continuous presence of *H pylori* resulting in the persistence of MALT in the gastric mucosa, which finally progress to form low- and

high-grade MALT lymphomas. MALT lymphomas are monoclonal proliferations of neoplastic B cells. It has the ability to infiltrate gastric glands. Gastric MALT lymphomas typically are low-grade T-cell–dependent B-cell lymphomas, and the antigenic stimulus for gastric MALT lymphomas is found to be *H pylori.*

The most deadlier complication of *H pylori* gastritis is the development of gastric carcinomas, especially in persons who develop extensive atrophy and intestinal metaplasia of the gastric mucosa. It is well accepted that a tedious process initiated by *H pylori* causing chronic inflammation in the gastric mucosa leading to chronic atrophic gastritis, intestinal metaplasia, dysplasia, and finally progressing to the development of adenocarcinoma.

Even though the relationship between *H pylori* and gastritis is constant, only a small proportion of people getting infected with *H pylori* develop gastric cancer. The incidence of gastric cancer usually parallels the incidence of *H pylori* infection in countries with a higher incidence of gastric cancer and is consistent with *H pylori* infection being the major cause of the precursor lesion, chronic atrophic gastritis.

Persistence of the organisms causing long-standing inflammation leads to accumulation of mutations in the gastric epithelial cells' genome, leads to an increased risk of malignant transformation and progression to

adenocarcinoma. Studies have proven that mutation in gastric epithelium is secondary to oxidative DNA damage which is associated with chronic inflammatory byproducts and also secondary to deficiency of DNA repair caused by chronic bacterial infection.[35][36][37]

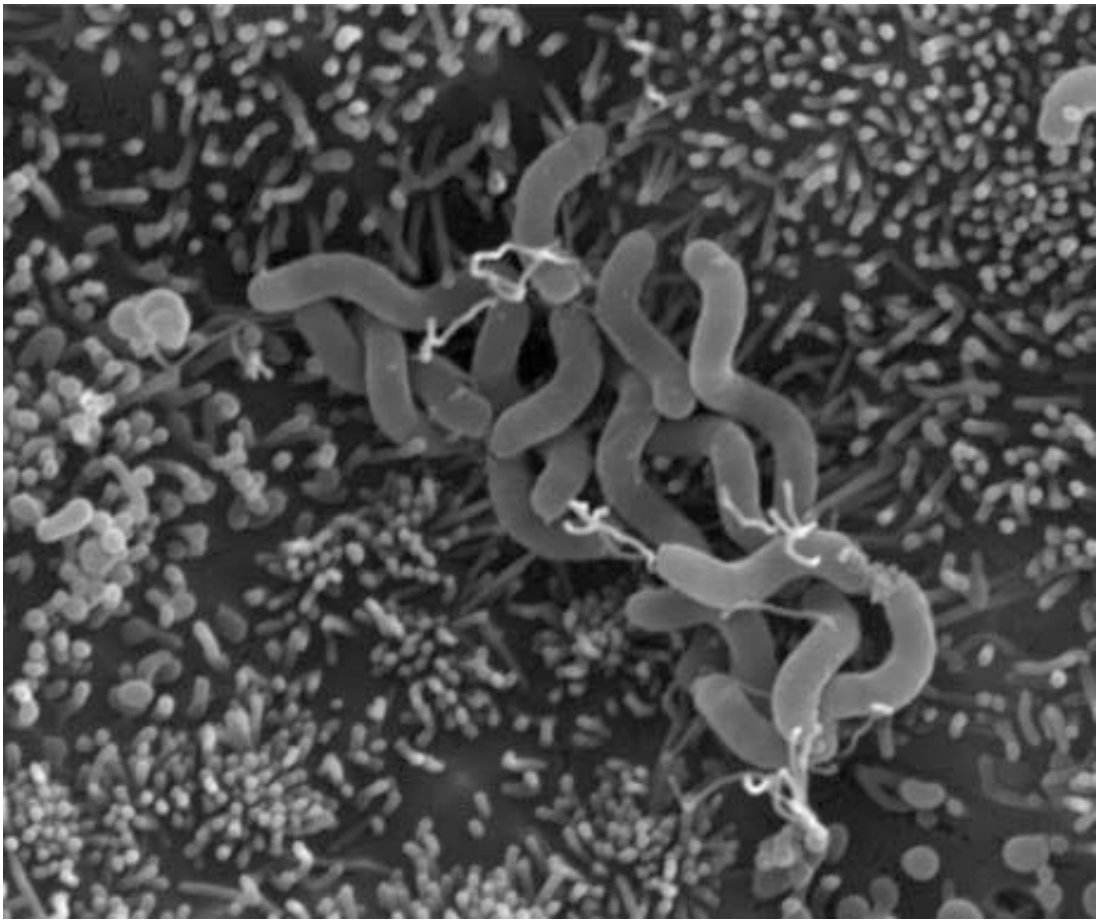
Even though the role of *H pylori* in peptic ulcer disease is well known, the role of the H. pylori infection in non-ulcer or functional dyspepsia still remains highly controversial. A recent meta-analysis proven that *H pylori* eradication therapy has associated with improvement in dyspeptic symptoms in patients who have functional dyspepsia in Asian, European, and American populations.

Although many study has illustrated that H *pylori* eradication may be beneficial for symptom relief for dyspepsia in some populations, routine *H pylori* testing and treatment in non ulcer dyspepsia is not currently widely accepted. Therefore, *H pylori* eradication strategies in patients with non ulcer dyspepsia should be considered on a patient-to-patient basis.

HELICOBACTER PYLORI

Helicobacter pylori is a spiral-shaped, microaerophilic, gram- negative bacterium. It was earlier called as Campylobacter pylori. It is one of the most common bacterial infection in the world as it infects about 50% of the population in some countries. It was first identified by scientists Barry Marshall and Robin Warren.

They found it in a person with chronic gastritis and gastric ulcers. It is also linked to the development of duodenal ulcers and gastric cancer and lymphoma. Hence the World Health Organisation has labelled the organism as a class 1 carcinogen. It invades the mucosal lining of the stomach and is found to be responsible for about 95% duodenal ulcer and about 75% gastric ulcer. However only 20% of the people infected with this organism remain symptomatic. It is also considered to play an important role in the normal gastric flora.[38][39]



H. PYLORI ELECTRON MICROSCOPIC IMAGE

Genome

H. pylori consists of a large diversity of strains, and hundreds of genomes have been completely sequenced. The genome of the strain "26695" consists of about 1.7 million base pairs, with some 1,576 genes. The pan-genome, that is a combined set of 30 sequenced strains, encodes 2,239 protein families (orthologous groups, OGs). Among them, 1248 OGs are conserved in all the 30 strains, and represent the universal core. The remaining 991 OGs correspond to the accessory genome in which 277 OGs are unique (i.e., OGs present in only one strain).

Transcriptome

In 2010, Sharma et al. presented a comprehensive analysis of transcription at single-nucleotide resolution by differential RNA-seq that confirmed the known acid induction of major virulence loci, such as the urease (ure) operon or the cag pathogenicity island. More importantly, this study identified a total of 1,907 transcriptional start sites, 337 primary operons, and 126 additional suboperons, and 66 monocistrons. Until 2010, only about 55 transcriptional start sites (TSSs) were known in this species. Notably, 27% of the primary TSSs are also antisense TSSs, indicating that similar to E. coli antisense transcription occurs across the entire H. pylori genome. At least one antisense TSS is associated with about 46% of all open reading frames, including many housekeeping genes. Most (about 50%) of the 5' UTRs are 20–40 nucleotides (nt) in length and support the AAGGag motif located about 6 nt (median distance) upstream of start codons as the consensus Shine–Dalgarno sequence in H. pylori.

Genes involved in virulence and pathogenesis

Study of the H. pylori genome is centered on attempts to understand pathogenesis, the ability of this organism to cause disease. About 29% of the loci have a colonization defect when mutated. Two of sequenced strains have an around 40-kb-long Cag pathogenicity island (a

common gene sequence believed responsible for pathogenesis) that contains over 40 genes. This pathogenicity island is usually absent from H. pylori strains isolated from humans who are carriers of H. pylori but remain asymptomatic.

The cagA gene codes for one of the major H. pylori virulence proteins. Bacterial strains with the cagA gene are associated with an ability to cause ulcers. The cagAgene codes for a relatively long (1186-amino acid) protein. The cag pathogenicity island (PAI) has about 30 genes, part of which code for a complex type IV secretion system. The low GC-content of the cag PAI relative to the rest of the Helicobacter genome suggests the island was acquired by horizontal transfer from another bacterial species.

Adaptation to the stomach's acidic environment

To avoid the acidic environment of the interior of the stomach (lumen), H. pylori uses its flagella to burrow into the mucus lining of the stomach to reach the epithelial cells underneath, where it is less acidic. H. pylori is able to sense the pH gradient in the mucus and move towards the less acidic region (chemotaxis). This also keeps the bacteria from being swept away into the lumen with the bacteria's mucus environment, which is constantly moving from its site of creation at the epithelium to its dissolution at the lumen interface.

H. pylori is found in the mucus, on the inner surface of the epithelium, and occasionally inside the epithelial cells themselves. It adheres to the epithelial cells by producing adhesins, which bind to lipids and carbohydrates in the epithelial cell membrane. One such adhesin, BabA, binds to the Lewis b antigen displayed on the surface of stomach epithelial cells. Another such adhesin, SabA, binds to increased levels of sialyl-Lewis x antigen expressed on gastric mucosa.

In addition to using chemotaxis to avoid areas of low pH, H. pylori also neutralizes the acid in its environment by producing large amounts of urease, which breaks down the urea present in the stomach to carbon dioxide and ammonia. These react with the strong acids in the environment to produce a neutralized area around H. pylori. Urease knockout mutants are incapable of colonization. In fact, urease expression is not only required for establishing initial colonization but also for maintaining chronic infection.

Cag pathogenicity Island

The pathogenicity of H. pylori may be increased by genes of the cag pathogenicity island; about 50–70% of H. pylori strains in Western countries carry it. Western people infected with strains carrying the cag PAI have a stronger inflammatory response in the stomach and are at a greater risk of developing peptic ulcers or stomach cancer than those

infected with strains lacking the island. Following attachment of H. pylori to stomach epithelial cells, the type IV secretion system expressed by the cag PAI "injects" the inflammation-inducing agent, peptidoglycan, from their own cell walls into the epithelial cells. The injected peptidoglycan is recognized by the cytoplasmic pattern recognition receptor (immune sensor) Nod1, which then stimulates expression of cytokines that promote inflammation.

The type-IV secretion apparatus also injects the cag PAI-encoded protein CagA into the stomach's epithelial cells, where it disrupts the cytoskeleton, adherence to adjacent cells, intracellular signaling, cell polarity, and other cellular activities. Once inside the cell, the CagA protein is phosphorylated on tyrosine residues by a host cell membrane-associated tyrosine kinase (TK). CagA then allosterically activates protein tyrosine phosphatase/protooncogene Shp2. Pathogenic strains of H. pylori have been shown to activate the epidermal growth factor receptor (EGFR), a membrane protein with a TK domain. Activation of the EGFR by H. pylori is associated with altered signal transduction and gene expression in host epithelial cells that may contribute to pathogenesis. A C-terminal region of the CagA protein (amino acids 873–1002) has also been suggested to be able to regulate host cell gene transcription, independent of protein tyrosine phosphorylation. A great

deal of diversity exists between strains of H. pylori, and the strain that infects a person can predict the outcome.

Transmission of H. pylori

H. pylori transmission by oral to oral and feco- oral are the most common routes. Crowded living conditions, poor sanitation, poor personal hygiene and a poor water supply are associated with higher rates of infection. The H. pylori uses its flagella to burrow in the stomach lining and reaches the epithelial cells, underneath which is less acidic. The H. pylori also survives in the highly acidic environment of the stomach by producing urease which converts the urea in the stomach to carbon di oxide and ammonia which acts as a buffer. This ammonia is toxic to human cells. The urease is also required for maintaining chronic infection.

Progression of H. pylori infection

The H. pylori affects the gastric mucosa by these slowly progressing steps[40] :

Stage 1 : Normal stomach lining (mucosa)

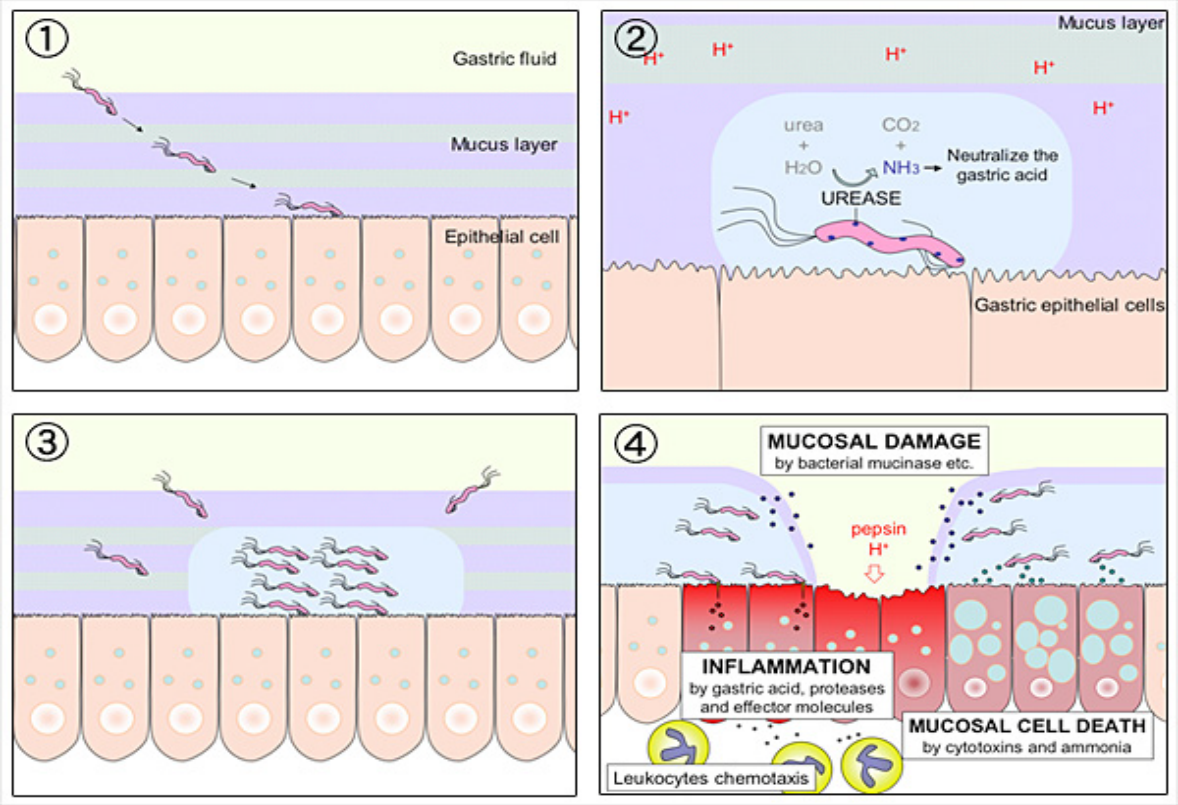
Stage 2 : Inflammation of the stomach lining (chronic gastritis)

Stage 3 : Loss of stomach cells and impaired digestive system (atrophic gastritis)

Stage 4 : Transformation of the stomach lining (intestinal metaplasia)

Stage 5 : Beginning stages of stomach cancer (dysplasia)

Stage 6 : Stomach cancer (gastric adenocarcinoma)



Progression of H. pylori

Diagnosis of H. pylori[41][42]

Tests for H. pylori are divided into invasive and non invasive tests:

Gastroduodenoscopy:

By endoscopy, biopsy is taken for histological examination and is considered to be gold standard. It has more than 90% sensitivity and specificity. In addition to identifying the presence of H. pylori the biopsy can also be used to assess the state of the gastric lining.

Urea breath test :

It is a rapid non-invasive test to detect the presence of active H.pylori infection. During a breath test, you swallow a pill, liquid or pudding that contains tagged carbon molecules. If you have an H. pylori infection, carbon is released, when the solution is broken down in your stomach.

Your body absorbs the carbon and expels it when you exhale. You exhale into a bag, and a special device detects the carbon molecules.

Acid-suppressing drugs known as proton pump inhibitors (PPIs), bismuth subsalicylate (Pepto-Bismol) and antibiotics can interfere with the accuracy of this test. So these medications need to be stopped for a week or two weeks before you have the test.

Serology

Antibodies for H. pylori are screened in the patient's blood. This test is not that accurate and cannot differentiate between current infection and recent exposure.

Stool H. pylori antigen test

It is an accurate test and is being used more frequently.

ENDOSCOPY

Flexible endoscope is the instrument of choice for endoscope due to its ease technically, acceptance by the patient and ability to reach up to the duodenum. Rigid endoscopes are rarely used nowadays, mainly for removing impacted and difficult foreign bodies in the oesophagus.

Preparation for Endoscopy

The patients should be kept on fasting for 4 to 8 hours depending upon the condition for which they are subjected to endoscopy. In case of obstructive lesions of the stomach, an early morning stomach wash may be helpful in making the stomach empty which is important for clear visualisation of the stomach and duodenum.

Procedure of Endoscopy

The patient is made to lie in left lateral position and the flexible oesophagoscope is introduced through the mouth and is manipulated to enter the esophagus. The esophagus is visualised all around and the lower oesophageal junction is given more importance. The oesophagoscope is then pushed into the stomach and the stomach is completely visualised. The oesophagoscope can be inserted further to visualise the duodenum. Throughout the course of procedure air and water can be flushed through the oesophagoscope for better visualisation. Also biopsy is done wherever required by inserting a flexible biopsy forceps through the oesophagoscope.

Biopsy sites:[43][44]

For chronic gastritis, the following recommendations, as per updated Sydney system classification are:

A. For optimal assessment, five biopsy specimens are taken, two from the antrum within 2 to 3 cm from the pylorus, one from the distal lesser curvature, and the other from the distal greater curvature, two from the corpus about 8 cm from the cardia (one from the lesser and the other from the greater curvature), and one from the incisura angularis.

B. Samples from antrum, corpus, and incisura angularis should be separately identifiable.

C. Transmission of information to the pathologist about the patient's endoscopic findings, clinical history, and biopsy sites is essential for successful clinicopathologic correlation in gastritis.

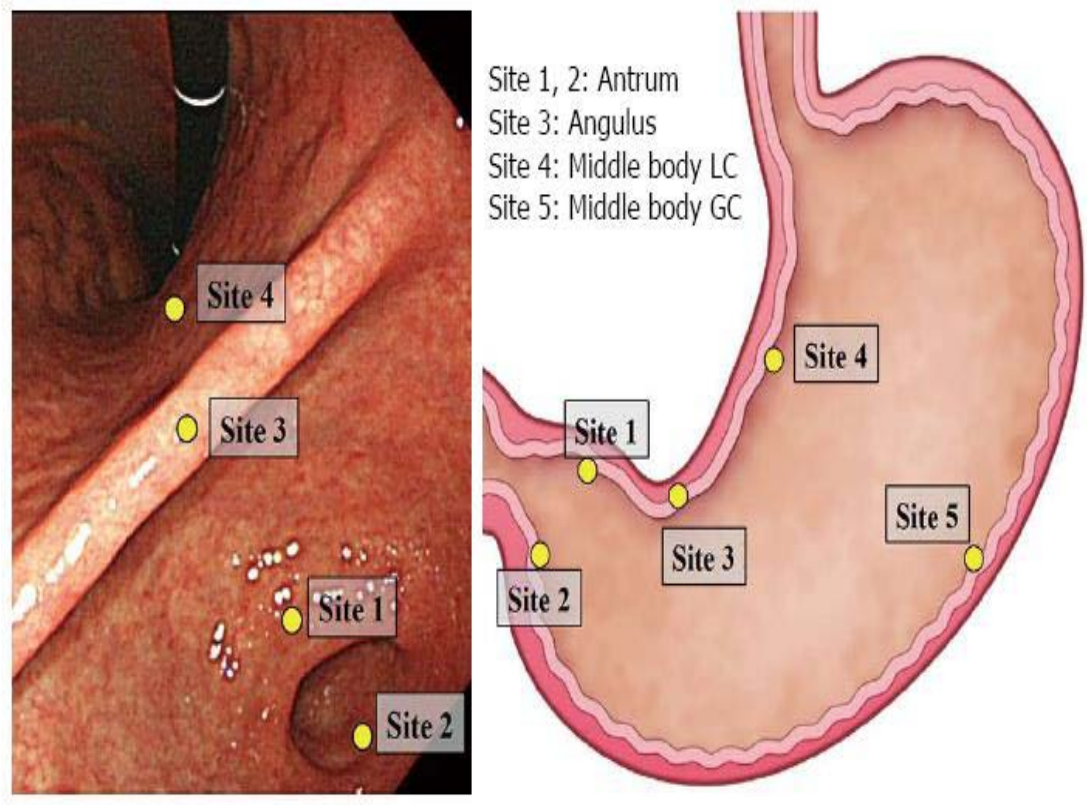
D. A special stain for H. pylori should be carried out before declaring an inflamed biopsy specimen negative.

E. An Alcian blue/Periodic acid schiff stain will facilitate the recognition of intestinal metaplasia.

Corpus biopsies are particularly valuable for yielding positive results after treatment, especially where proton pump inhibitors have been used.

Maximal degrees of gastric mucosal atrophy and intestinal metaplasia are consistently found in the region of the incisura angularis, which is also the site most likely to reveal premalignant dysplasia.

In addition to usual eosin and haematoxylin stain which can show chronic gastritis, additional stains like modified Giemsa, Warthin-Starry, or the new Genta stain are used to identify H. pylori.



Biopsy sites : Recommended Updated Sydney grading system

Complications of Endoscopy

Cardiovascular complications including angina, arrhythmias, and stroke

Aspiration pneumonia

Infection

Bleeding

Perforation (in the pharynx or oesophagus, may follow biopsy of oesophageal or gastric malignancy)

Minor throat and abdominal discomfort

Macroscopic findings in endoscopy

The *H pylori* – infected gastritis shows a range of findings, best demonstrated during endoscopic examination. There may be erythema, a nodular appearance of the mucosa, frank ulceration, or the mucosa may appear relatively unremarkable. However, the endoscopic findings are not specific, and, therefore, the standard for diagnosis remains the evaluation of gastric mucosa biopsies.

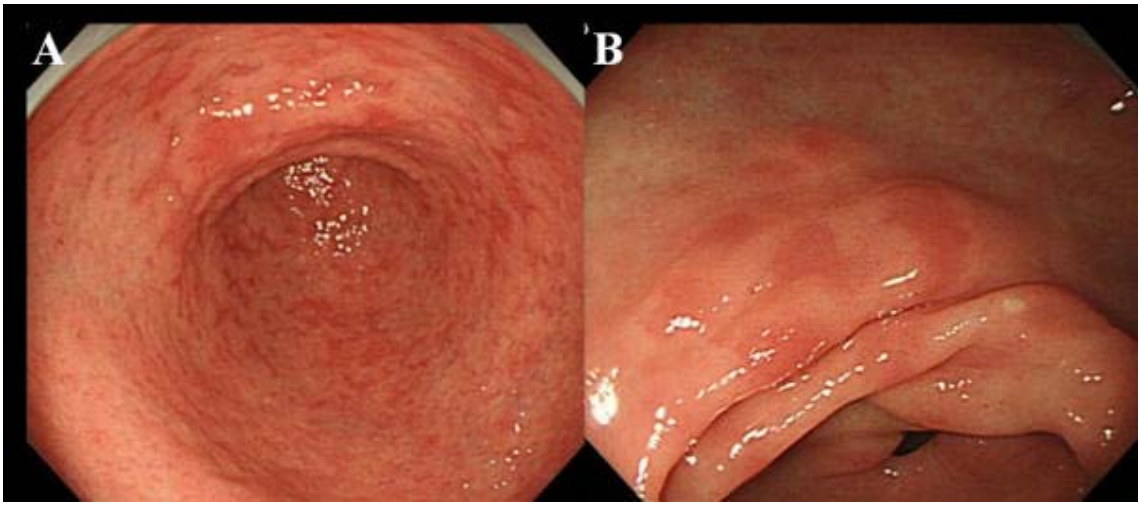
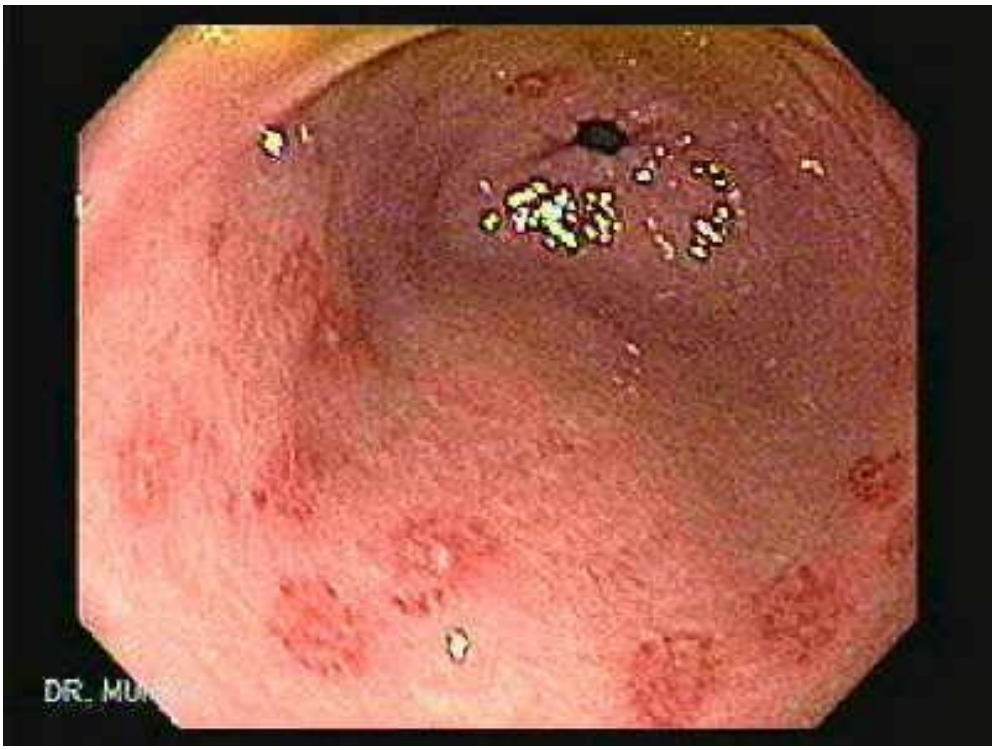


Figure showing multiple flat erythema and nodules in antrum and lesser curvature.



Gastric antrum showing multiple erythematous antral region

Biopsy from antral nodularity of gastric mucosa

Microscopic (histologic) description[45][46]

1. Bacteria is curved, spirochete-like, found in superficial mucus layer and also along the microvilli of epithelial cells.

2. Invasion is unusual, proton pump inhibitor use may increase risk of invasion. Usually not seen in areas of intestinal metaplasia.

3. Associated with chronic inflammatory infiltrate with germinal centers (follicular gastritis) and plasma cells in lamina propria.

4. Active inflammation is described if neutrophils are in glandular or surface epithelial layer. Presence of active inflammation after eradication therapy is a sign of treatment failure

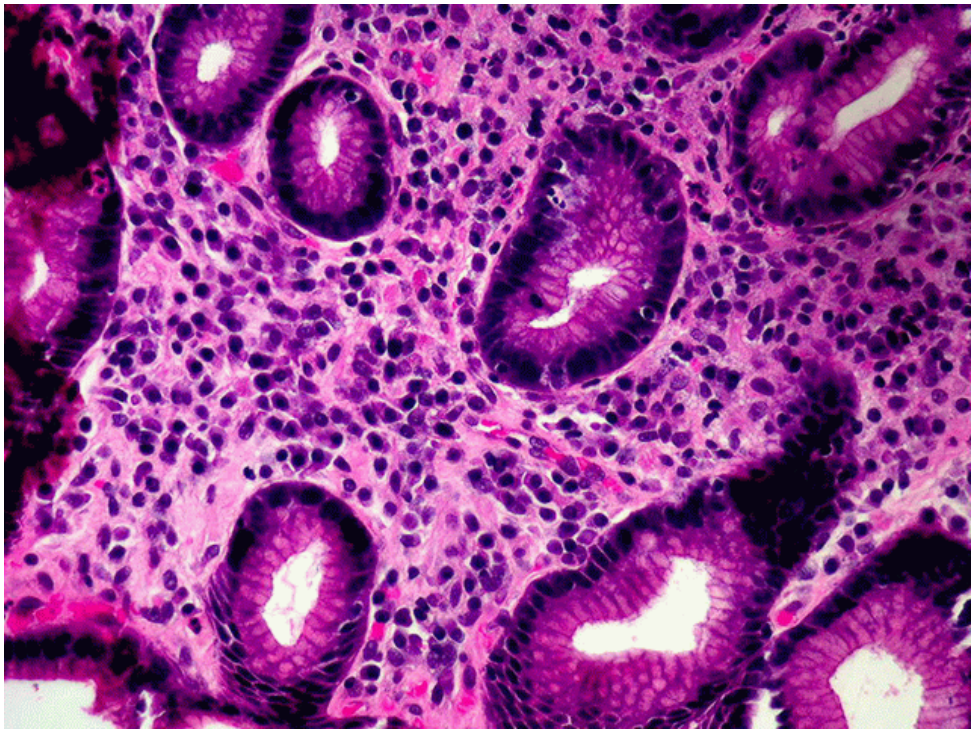
5. Antibiotics may cause *H. pylori* to assume coccoid appearance.

6. Presence of follicles is strongly associated with *H. pylori*, the density of follicles is highest in the angulus, the most common site of gastric lymphoma. The lowest density of follicles is in the proximal greater curvature, where incidence of *H. pylori* induced gastric lymphoma is lowest.

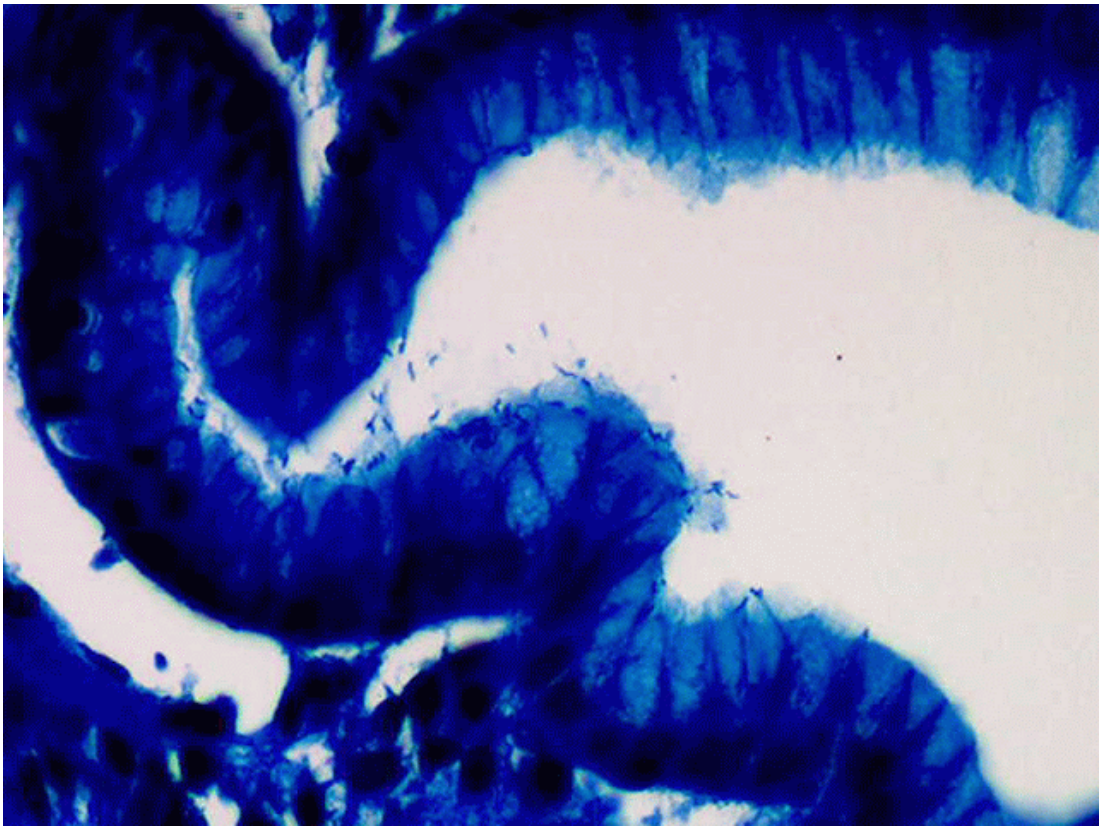
7. Chronic proton inhibitor use without antibiotics leads to relatively decreased inflammation in the antrum and increased inflammation in the body with decreased numbers of microorganisms.

8. Regenerative change: enlarged, hyperchromatic nuclei in surface epithelial cells, with diminished mucus vacuoles and frequent mitotic figures.

9. Acute infection associated with erosions, ulcers, hemorrhage.



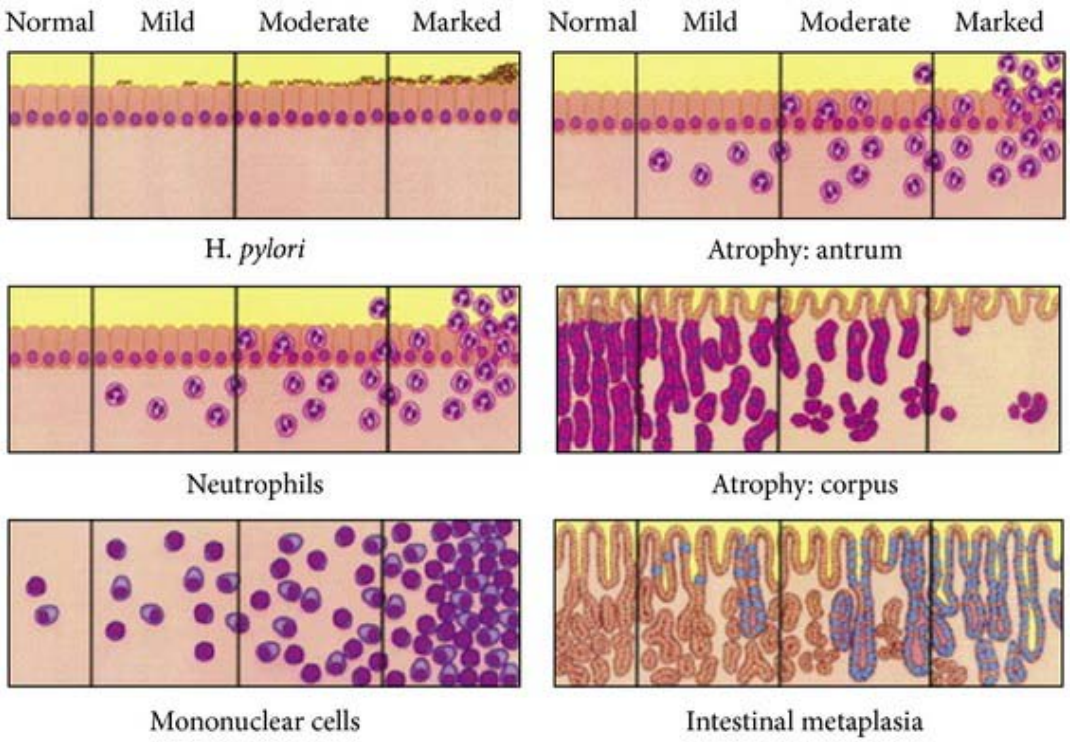
The antrum of the stomach shows chronic gastritis with lymphocytes, plasma cells, and rare eosinophils in the lamina propria.



Helicobacter pylori gastritis (high resolution)

Rod-shaped organisms are present along the luminal surfaces of the epithelium and in the luminal mucus. They do not invade the mucosa. The bacteria are small and in H&E stain have a pale eosinophilic appearance. They are best seen on giemsa stain, where they stain bluish-purple.[51][52]

Updated Sydney system of grading gastritis: A visual scale



Histopathology Grading of Gastritis

All the obtained biopsies are collected and placed on filter paper, fixed in 10% neutral formalin. It is sent for preparation of formalin-fixed, paraffin-embedded tissue blocks. Three-micrometer-thick sections are prepared. One set of tissue sections is stained with eosin and hematoxylin and the other with Giemsa stain for histopathological examination (by 2 experienced pathologists), including detection of H. pylori in the gastric mucosa[47].

The biopsies are looked for the intensity of mononuclear inflammatory cellular infiltrates, inflammatory activity (neutrophilic infiltrations), glandular atrophy, metaplasia, reparative atypia, and dysplasia. Additionally, the cases are graded according to the Houston-updated Sydney system grading for gastritis, which is graded according to the intensity of mononuclear inflammatory cellular infiltrates within the lamina propria: absent inflammation (Grade 0), mild inflammation (Grade 1), moderate inflammation (Grade 2), and severe inflammation (Grade 3)[48]

Histopathology Grading of Gastritis

		Corpus			
		No atrophy (grade 0)	Mild atrophy (grade 1)	Moderate atrophy (grade 2)	Severe atrophy (grade 3)
Antrum	No atrophy (grade 0)	Stage 0	Stage I	Stage II	Stage II
	Mild atrophy (grade 1)	Stage I	Stage I	Stage II	Stage III
	Moderate atrophy (grade 2)	Stage II	Stage II	Stage III	Stage IV
	Severe atrophy (grade 3)	Stage III	Stage III	Stage IV	Stage IV

Treatment for H. pylori infection:[49][50]

First line therapy:

First-line therapy has been used for *H. pylori* eradication in many parts of the world. It consists of a triple therapy using a Proton pump inhibitor or ranitidine bismuth citrate, combined with clarithromycin and amoxicillin or metronidazole for those individuals with penicillin allergy. All are given twice daily. However, even with correct use of these combinations, infection is not eradicated in 10-23% of patients. The recommended duration of treatment range is between 7 and 14 days. The emergence of drug resistance and decreasing drug efficacy, has made the second-line therapy necessary.

Second line therapy:

H. pylori may acquire resistance by acquisition of mutations and recombination of genes from other bacteria. Chromosomic point mutations can also induce resistance. Metronidazole targets DNA and has a high mutation rate.. Clarithromycin and Metronidazole are two antibiotics noted for resistance and most of *H. pylori* isolates after two eradication failures are found to be resistant to the two drugs mentioned above. Subsequently, quadruple therapy which consists of PPI, bismuth, metronidazole and tetracycline is a recommended alternative to first-line

treatment, which are used in areas of high antibiotic resistance. Where bismuth is not available, second-line therapy may be with PPI-based triple therapy.

Third line therapy or salvage therapy:

This is given after multiple (at least two) treatment failures with different regimens. Basically, it would be chosen based on the results of antimicrobial susceptibility testing. Often, a careful review of agents used previously will enable a regimen to be identified that will be successful. It was found that most of *H. pylori* isolates after two eradication failures are resistant to metronidazole and clarithromycin. Therefore, it is recommended that these two drugs should be removed from the third-line therapy. These third-line therapies are the new emerging therapies.

Levofloxacin based, Rifabutin and Rifampicin based, Furazolidone based, Doxycycline based and Lactoferrin based therapies are under study.

PARAMETERS TO BE EVALUATED:

Endoscopic features of chronic gastritis:

Erythema, hyperemia, atrophy, and mucosal nodularity are the features according to the criteria of the Sydney grading system.

Histologic features of chronic gastritis:

Increased lymphocytes and plasma cells in the lamina propria are the features according to the criteria of Sydney grading system.

Histologic features of H. pylori:

1. Comma or S shaped bacilli (2-4 μm long and 0.5-1 μm thick).
2. Bacilli adhered on cell surface or free in mucosa layer, with its typical morphology, and forming at least small colonies.
3. Isolated forms of H. pylori like bacterium are considered negative by histology.

MATERIALS AND METHODS

Study population:

This consists of patients with complaints of dyspepsia for more than 6 months at General surgery department, Govt Kilpauk Medical College and Hospital, Chennai.

Method of collection of data (including sampling procedure):

A. Study design: Cross sectional study

B. Place of study: Govt.Kilpauk Medical College and Hospital, Chennai.

C. Study sample size: **87**

N = 4pq/d^2= 4*85*15/ (7.65)^2**= *87***

p: Prevalence of H. pylori in chronic gastritis (85%)

q: 100-p (100-85= 15%)

d: allowable relative error 9% (9*85/100 =7.65 %)

Confidence level **–** 95%

D. Study period: December 2016 **–** September 2017

E. Method of sampling: Random sampling.

F. Inclusion criteria:

1. Patients between the ages of 20 – 60 years
2. Symptomatic patients with epigastric pain, postprandial fullness, heartburns, bloating, belching, nausea, vomiting for minimal period of 6 months.
3. Patients were given written informed consent

G. Exclusion criteria:

H.pylori regimen before 4 weeks of endoscopy.

Congestive cardiac failure

MI or chest pain within last 12 months

COPD (FEV less than 1.25, home oxygen use)

Coagulopathy (INR greater than 2) or bleeding disorders

Platelet count less than 75,000 cells/cu.mm

H. Methodology:

1. **Sample:** Patients will be selected on basis of inclusion and exclusion criteria.
2. **Written informed consent** will be taken (Attached below).

3. **PROFORMA:**

1. Patient name:

2. IP No:

3. Department:

4. Hospital:

5. Age:

6. Sex:

7. Address:

8. Contact No:

9. Occupation:

10. Chief complaints:

11. Past history:

12. General examination:

13. Vitals:

14. Abdominal examination:

15. Cardiovascular and Respiratory system examination:

16. Investigations:

Complete hemogram

Renal function tests and Electrolytes

Liver function test

BT/CT

VCTC/HbSAg

Chest X-ray and Abdomen X-ray

17. Specific investigations:

USG Abdomen and pelvis

Echocardiography

Endoscopic biopsies

5 biopsies taken from following sites:

1. 1 from antrum 2-3 cm from pylorus lesser curvature,
2. 1 from antrum 2-3 cm from the pylorus greater curvature,
3. 1 from the corpus, 8cm from the cardia lesser curvature,
4. 1 from the corpus 8cm from the cardia greater curvature,
5. 1 from the angularis.

Histopathology examination:

Biopsied specimen are sent for histopathology examination by preserving in 10% buffered formalin solution.

DATA ANALYSIS

Data collection was done. All the values were entered in Microsoft excel sheet. The values were used for analysis.

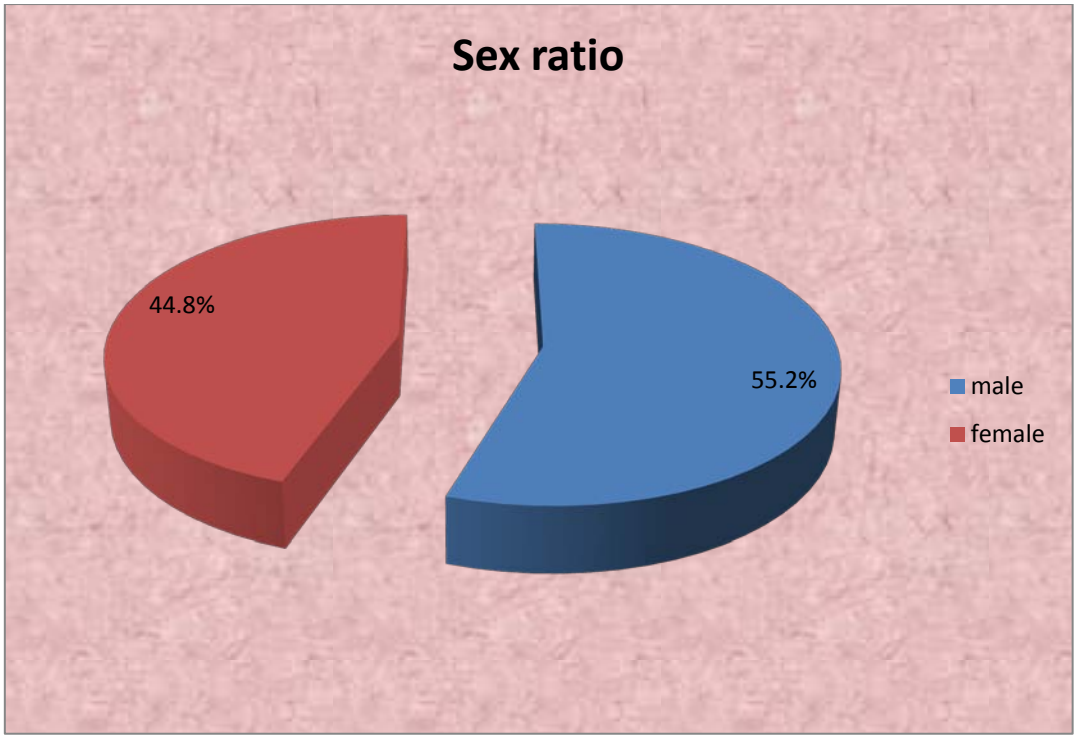
Chi square test was used to analyze the values collected. The software tools used for the purpose were downloaded from the internet. The values obtained were confirmed using another software to check the validity.

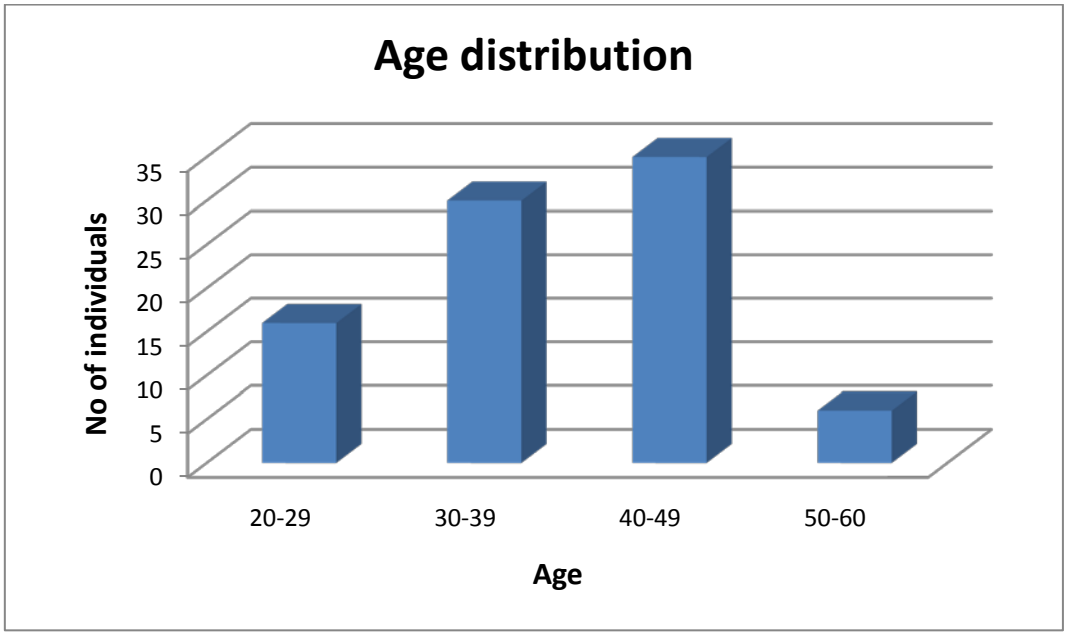
The mean, standard deviation, standard error of mean and the p-value were calculated.

The results were again converted into tables, pie charts, donut charts and bar diagrams and were presented as follows.

Socio demographic profile of study population

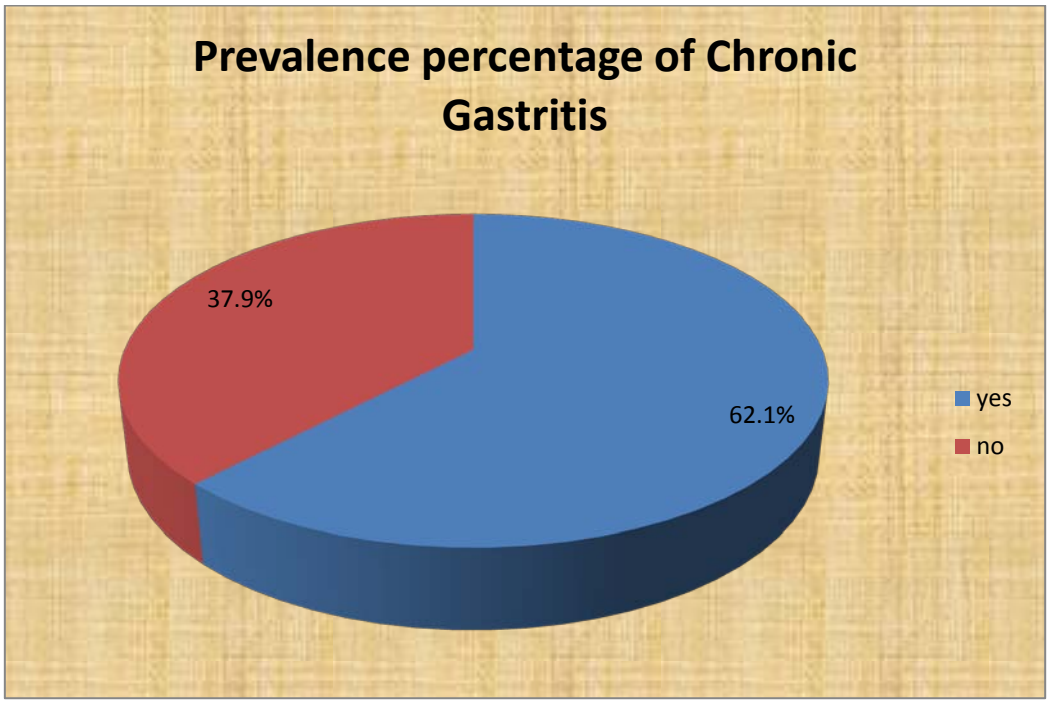
Parameter	Frequency	Percentage (%)
Age		
20-29 yrs	16	18.4
30-39 yrs	30	34.5
40-49 yrs	35	40.2
50- 60 yrs	6	6.9
Gender		
Female	39	44.8
Male	48	55.2
Smoking		
Yes	31	35.6
No	56	64.4
Alcohol		
Yes	30	34.5
No	57	65.5
Tobacco use		
Yes	38	43.7
No	49	56.3
Diet		
Veg	5	5.7
Non veg	82	94.3

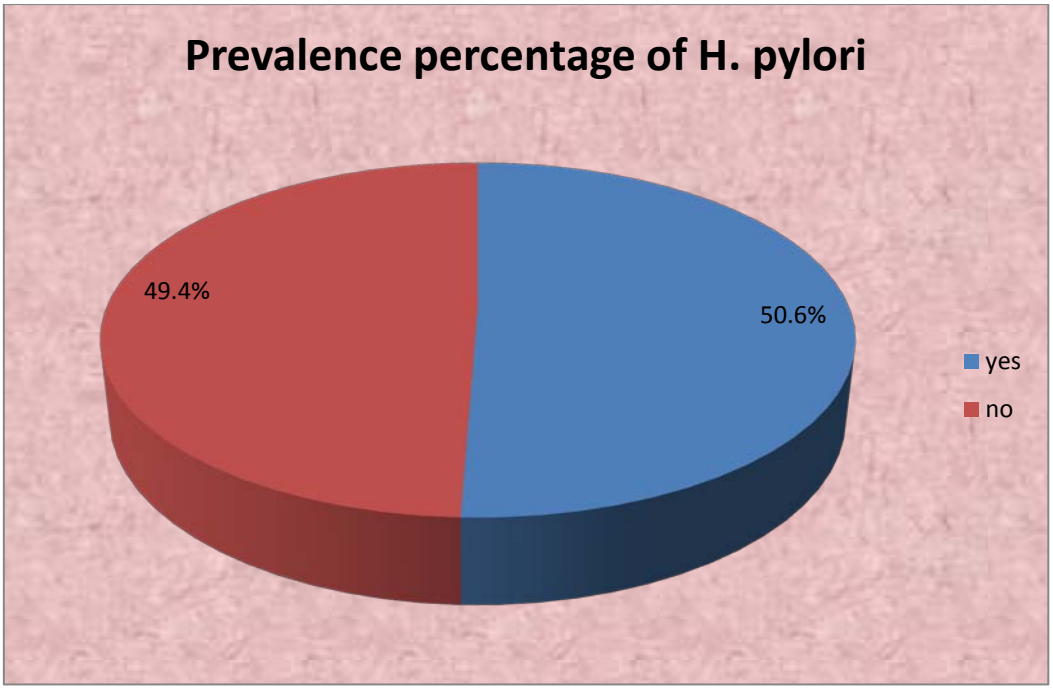

Sex ratio
44.8%
55.2%
male
female


Age distribution
No of individuals
35
30
25
20
15
10
5
0
20-29
30-39
40-49
50-60
Age

Prevalence of chronic gastritis and H. pylori among study population

Parameter	Frequency	Percentage (%)
Chronic gastritis		
Yes	54	62.1
No	33	37.9
H.pylori		
Yes	44	50.6
No	43	49.4

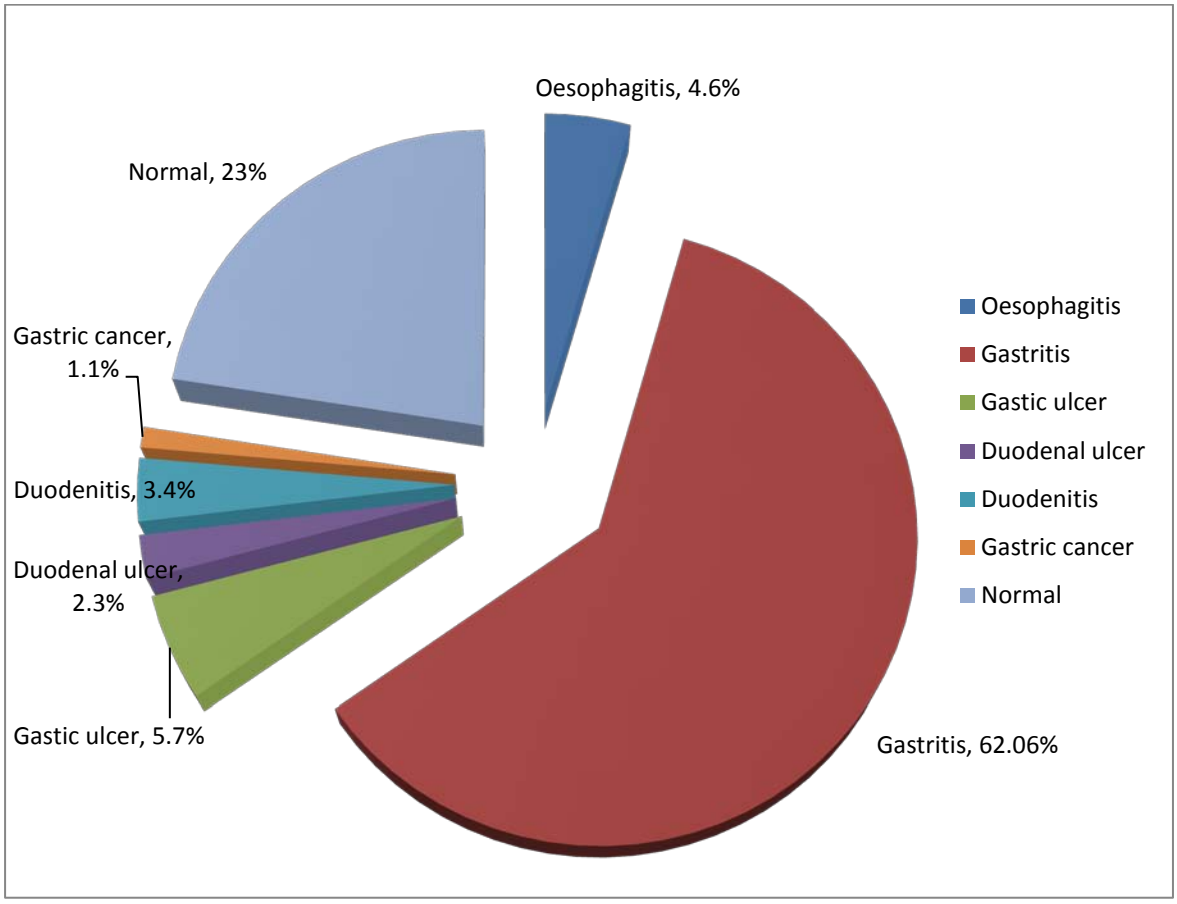


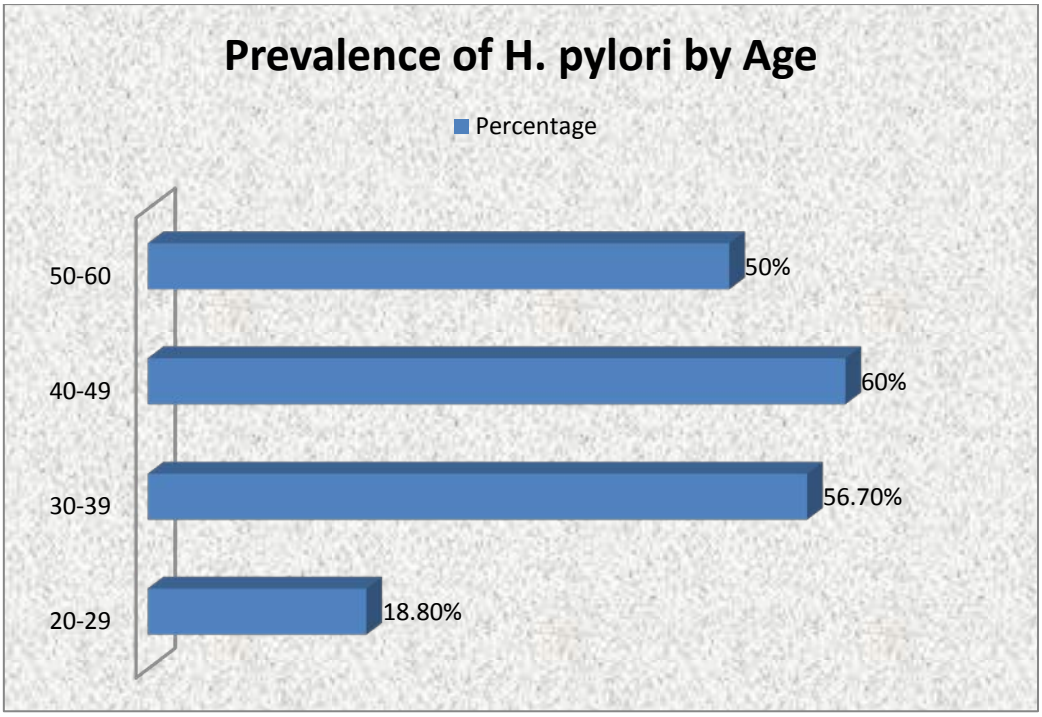


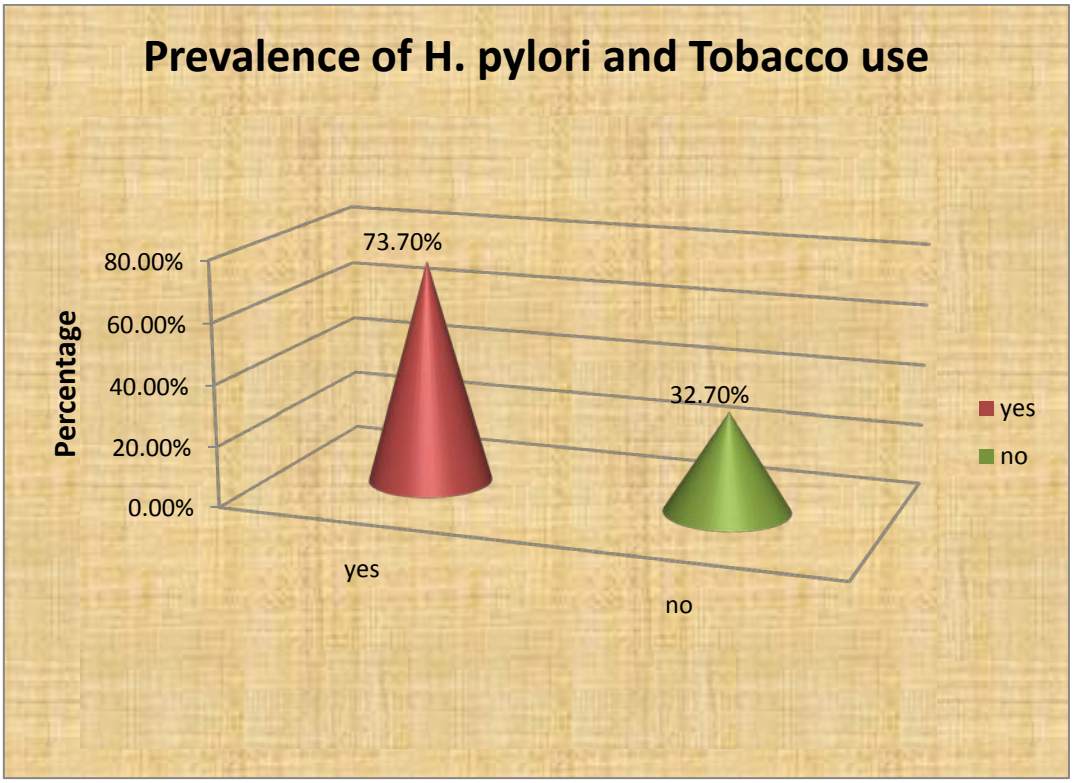
Endoscopic finding among study population

Endoscopic finding	Frequency	Percentage (%)
Oesophagitis	4	4.6
Gastritis	54	62.06
Gastric ulcer	5	5.7
Duodenal ulcer	2	2.3
Duodenitis	3	3.4
Gastric cancer	1	1.1
Normal	20	23.0

Endoscopic findings in dyspeptic patients







Association between H. Pylori and certain variables

Parameters	H. Pylori N (%)	Chi square	P-value
Age			
20-29 yrs	3(18.8)	8.173	0.043
30-39 yrs	17(56.7)		
40-49 yrs	21(60)		
50- 60 yrs	3(50)		
Gender			
Female	20(51.3)	0.014	0.905
Male	24(50)		
Smoking			
Yes	19(61.3)	2.21	0.137
No	25(44.6)		
Alcohol			
Yes	19(63.3)	2.98	0.084
No	25(43.9)		
Tobacco use			
Yes	28(73.7)	14.4	0.000
No	16(32.7)		
Diet			
Veg	42(51.2)	0.237	0.626
Non veg	2(40)		
Chronic gastritis			
Yes	38(70.4)	22.318	0.000
No	6(18.2)		

RESULTS

Of the 87 patients in study population, greater proportion of study participants were in age group 40-49 yrs. The majority of the study participants (55.2%) were males. In this study, nearly 35% had history of smoking and 35% had history of alcohol intake. About 43% of study participants were tobacco users. Majority of the study participants (94.3%) were non vegetarians.

In this study 62% of participants had chronic gastritis.

H.pylori was present among 50.6% of study participants.

The most common endoscopic finding is gastritis (62.06%). Other less common findings were oesophagitis (4.6%), gastric ulcer (5.7%), duodenitis (3.4%), duodenal ulcer (2.3%) and gastric cancer (1.1%). For 23% of patients the endoscopic finding was normal.

The prevalence of H.Pylori was maximum in 40- 49 years age group (60%) and minimum in 20-29 years age group.

The prevalence of H.Pylori was high in tobacco users (73.7%) and less in non tobacco users (32.7%).

The prevalence of H.Pylori was maximum in 40- 49 years age group (60%) and minimum in 20-29 years age group. The p value <0.05

is considered as statistically significant. This difference was statistically significant (p=0.043). No statistical association found between gender, smoking, alcohol, diet and H.Pylori prevalence. The prevalence of H.Pylori was high in tobacco users (73.7%) and less in non tobacco users (32.7%). This difference was statistically significant (p=0.000). The prevalence of H.Pylori was high in patients with chronic gastritis (70.4%) and less in patients without gastritis (18.2%). This difference was statistically significant (p=0.000).

DISCUSSION

H. pylori is a gram-negative, microaerophilic bacterium that can inhabit various areas of the stomach and duodenum. It causes a chronic low-level inflammation of the stomach lining, and is strongly linked to the development of duodenal and gastric ulcers.

The diagnosis of *H. pylori* by culture, gram stain and histology, which are biopsy based methods, is well established. In developing countries like India, problems associated with histological diagnosis of *H. pylori* arise because the result depend on the competence of the pathologist, the time spent to examine the slides (inter-observer variability) and the variability of staining techniques.15 Special stains for biopsy specimens improve visual detection of the bacteria. To mitigate these problems in our study, the service of a Gastrointestinal Pathologist was employed and Giemsa stain was used in addition to routine H&E.

There was no gender inequality in the distribution of H. pylori i.e. 50% of the male study population were infected with H. pylori and 51% of female population were infected with H. pylori, which is in consistent with Adlekha et al[5], study conducted in a patients undergoing upper gastrointestinal scopy, but goes against Nam SY et al[25], who said the prevalence is more among male than female.

Similarly study done by Tarkashveili et al[6], Khan AR et al[22], Joutei HA[19], Fraser AG[15] all supports that there is no gender inequality for the prevalence of H. pylori

In our study conducted, the most common age group infected with H. pylori is 40-49 years of age. On comparing this results with Baako BM et al[12], Hashemi et al[14], Fraser AG et al[15], it was found that it is in consistence with their studies. In our study, 60%, 56.6%, 50%, and 18.8% of individuals in age group 40-49, 30-39, 50-60, 20-29 years of age were infected with H. pylori respectively.

In our study among the 87 dyspeptic patients, 23% of the patients had normal gastric mucosa during endoscopy, 62.06% of patients had gastritis, 5.7% of the patients had gastric ulcer and 2.3% of patients had duodenal ulcer, 3.4% of individuals had duodenitis and 1.1% individual had gastric cancer. It has been seen in our study that the most common endoscopic findings in dyspeptic patients is gastritis amounting up to 62%. It was consistent with the studies conducted by Adhlekha et al[5], Abiodun Christopher et al[7], Hashemi MR et al[14], Al Akwaa AM et al[17], Oltega JB et al[18] and Alsaimary et al[20]. The correlation of endoscopic abnormality with *H. pylori* infection was statistically highly significant with a $P < 0.01$, proving endoscopic changes to be a sensitive indicator of *H. pylori* infection. This is in contrast to the observation laid

by Ozdil et al[26], in which the correlation was not statistically significant.

In our study population, the prevalence of chronic gastritis was found to be 62% and it is consistent with Nguyen et al[21], Mbengue M et al[10] who also found the prevalence between 60-80%

In our study population almost 50% of individuals are infected with H. pylori, which is consistent with Adlekha et al[5].

The prevalence of H. pylori among tobacco cheweres was 73.7% and it was found to be statistically significant which was similar in the study conducted by Ortega JP[18]

The prevalence of H. pylori among smokers, alcoholic and non vegetarian diets were not significant statistically and was in consistent with Fraser AG et al[15].

The prevalence of H. pylori in chronic gastritis in our study population is 70.4% and it is statistically significant and in concordance with the studies conducted by Adlekha S et al[5]. Tarkasvili et al[6], Abiodun Christopher et al[7], Oluwasola et al[8], Ogutu EO et al[9], Baako BN et al[12], Al Akwaa AM et al[17].

CONCLUSION

The study was done with the objective to find the prevalence of H. pylori in chronic gastritis.

The study showed that 62.1% of study population were having chronic gastritis and 50.6% of individuals are infected with H. pylori.

It also shown that 70.4% of individuals in chronic gastritis are affected with H. pylori.

This study shows the significant relationship between chronic gastritis and H. pylori infection.

LIMITATIONS

The present study has a major limitation that association of *H. pylori* infection with life-style related modifiable factors was not accessed. There is a need of another broader study in this region, assessing the association of different demographic and life-style factors and pre-existing conditions like diabetes mellitus with prevalence of *H. pylori* infection and follow-up of the patients after treatment and life-style modifications.

BIBLIOGRAPHY

1. Munnangi S. and Sonnenberg A. Time Trends of Physician Visits and Treatment Patterns of Peptic Ulcer Disease in the United States. *Arch Intern Med.* vol. 175, July 14, 1997. pp 1489-94.

2. *Helicobacter pylori* in Peptic Ulcer Disease, National Institutes of Health Consensus Development Panel on *Helicobacter pylori* in Peptic Ulcer Disease, *Journal of the American Medical Association*. Volume 272, no. 1. July 6, 1994, pp 65-69.

3. Eker R. Carcinomas of the stomach: investigation of the lymphatic spread from gastric carcinomas after totle and partial gastrectomy. Acta Chir Scand 1951;101: 112–26.

4. Latarjet A, Wertheimer P. L'énervation gastrique. Données expérimentales. Déductions cliniques. J Méd Lyon 1921;36: 1289–302.

5. Adlekha, S et al. "Prevalence of *Helicobacter Pylori* Infection Among Patients Undergoing Upper Gastrointestinal Endoscopy in a Medical College Hospital in Kerala, India." *Annals of Medical and Health Sciences Research* 3.4 (2013): 559–563. *PMC*. Web. 10 Sept. 2017.

6. Tarkhashvili, Nato et al. “*Helicobacter Pylori* Infection in Patients Undergoing Upper Endoscopy, Republic of Georgia.” *Emerging Infectious Diseases* 15.3 (2009): 504–505. *PMC*. Web. 10 Sept. 2017.

7. Abiodun Christopher, Jemilohun et al. “Prevalence of Helicobacter Pylori among Nigerian Patients with Dyspepsia in Ibadan.” *The Pan African Medical Journal* 6 (2010): 18. Print.

8. Oluwasola AO, Ogunbiyi JO. Chronic gastritis and Helicobacter pylori infection in University College Hospital Ibadan, Nigeria--a study of 85 fibre optic gastric biopsies. Niger J Med. 2004 Oct-Dec;13(4):372-8. PubMed PMID: 15523864.

9. Ogutu EO, Kang'ethe SK, Nyabola L, Nyong'o A. Endoscopic findings and prevalence of Helicobacter pylori in Kenyan patients with dyspepsia. East Afr MedJ. 1998 Feb;75(2):85-9. PubMed PMID: 9640829.

10. Mbengue M, Diouf ML, Dangou JM, Ka MM, Ba-Seck A, Ndiaye MF, Moreira-DiopT,Ndiaye PD, Bao O. [Frequency of Helicobacter pylori infection in symptomaticpatients in Senegal]. Med Trop (Mars). 1997;57(3):256-8. French. PubMed PMID: 9513152.

11. Aduful, HK et al. “Upper Gastrointestinal Endoscopy at the Korle Bu Teaching Hospital, Accra, Ghana.” *Ghana Medical Journal* 41.1 (2007): 12–16. Print.

12. Baako BN, Darko R. Incidence of Helicobacter pylori infection in Ghanaian patients with dyspeptic symptoms referred for upper gastrointestinal endoscopy. West Afr J Med. 1996 Oct-Dec;15(4):223-7. PubMed PMID: 9020601.

13. Xia B, Xia HH, Ma CW, Wong KW, Fung FM, Hui CK, Chan CK, Chan AO, Lai KC, Yuen MF, Wong BC. Trends in the prevalence of peptic ulcer disease and Helicobacter pylori infection in family physician-referred uninvestigated dyspeptic patients in Hong Kong. Aliment PharmacolTher. 2005 Aug 1;22(3):243-9. PubMed PMID:16091062.

14. Hashemi MR, Rahnavardi M, Bikdeli B, DehghaniZahedani M. H pylori infection among 1000 southern Iranian dyspeptic patients. World J Gastroenterol. 2006 Sep 14;12(34):5479-82. PubMed PMID: 17006984; PubMed Central PMCID: PMC4088229.

15. Fraser AG, Scragg R, Metcalf P, McCullough S, Yeates NJ. Prevalence of Helicobacter pylori infection in different ethnic

groups in New Zealand children and adults. Aust N Z J Med. 1996 Oct;26(5):646-51. PubMed PMID: 8958359.

16. Nakajima S, Nishiyama Y, Yamaoka M, Yasuoka T, Cho E. Changes in the prevalence of Helicobacter pylori infection and gastrointestinal diseases in the past 17 years. J GastroenterolHepatol. 2010 May;25Suppl 1:S99-S110. doi: 10.1111/j.1440-1746.2009.06214.x. PubMed PMID: 20586876.

17. Al-Akwaa AM. Prevalence of *Helicobacter pylori* Infection in a Group of Morbidly Obese Saudi Patients undergoing Bariatric Surgery: A Preliminary Report. *Saudi Journal of Gastroenterology : Official Journal of the Saudi Gastroenterology Association*. 2010;16(4):264-267. doi:10.4103/1319-3767.70610.

18. Ortega JP, Espino A, Calvo B A, Verdugo P, Pruyas M, Nilsen E, Villarroe L, Padilla O, Riquelme A, Rollán A. [Helicobacter pylori infection in symptomatic patients with benign gastroduodenal diseases: analysis of 5.664 cases]. Rev Med Chil. 2010 May;138(5):529-35. doi: /S0034-98872010000500001. Epub 2010 Jul 12. Spanish. PubMed PMID: 20668806.

19. Joutei HA, Hilali A, Fechtali T, Rhallabi N, Benomar H. [Helicobacter pylori infection in 755 patients with digestive

complaints: Pasteur Institute, Morocco, 1998-2007]. East Mediterr Health J. 2010 Jul;16(7):778-82. French. PubMed PMID: 20799536.

20. Alsaimary, Ihsan et al. “Clinical Findings and Prevalence of Helicobacter Pylori in Patients with Gastritis B in Al-Basrah Governorate.” *Oman Medical Journal*24.3 (2009): 208–211. *PMC*. Web. 15 Sept. 2017.

21. Nguyen, Tung L et al. “*Helicobacter Pylori* infection and Gastroduodenal Diseases in Vietnam: A Cross-Sectional, Hospital-Based Study.” *BMC Gastroenterology* 10 (2010): 114. *PMC*. Web. 15 Sept. 2017.

22. Khan AR. An age- and gender-specific analysis of H. Pylori infection. Ann Saudi Med. 1998 Jan-Feb;18(1):6-8. PubMed PMID: 17341905.

23. Gong YH, Sun LP, Jin SG, Yuan Y. Comparative study of serology and histology based detection of *Helicobacter pylori* infections: a large population-based study of 7,241 subjects from China. Eur J Clin Microbiol Infect Dis. 2010;29:907–11.

24. Li Z, Zou D, Ma X, Chen J, Shi X, Gong Y, Man X, et al. Epidemiology of peptic ulcer disease: endoscopic results of the

systematic investigation of gastrointestinal disease in China. Am J Gastroenterol. 2010;105:2570–7.

25. Nam SY, Choi IJ, Ryu KH, Kim BC, Kim CG, Nam BH. Effect of *Helicobacter pylori* infection and its eradication on reflux esophagitis and reflux symptoms. Am J Gastroenterol. 2010;105:2153–62.

26. Ozdil K, Sahin A, Kahraman R, Yuzbasioglu B, Demirdag H, Calhan T, et al. Current prevalence of intestinal metaplasia and *Helicobacter pylori* infection in dyspeptic adult patients from Turkey. Hepatogastroenterology. 2010;57:1563–6.

27. Tsukanov VV, Butorin NN, Maady AS, Shtygasheva OV, Amelchugova OS, Tonkikh JL, et al. *Helicobacter pylori* Infection, Intestinal Metaplasia, and Gastric Cancer Risk in Eastern Siberia. Helicobacter. 2011;16:107–12.

28. Ullah SS, Shamsuzzaman SM, Ara MN, Islam MN, Islam MS, Saha M, et al. Seropositivity of *Helicobacter Pylori* among the fish handlers. Mymensingh Med J. 2010;19:219–24.

29. Rahim AA, Lee YY, Majid NA, Choo KE, Raj SM, Derakhshan MH, et al.*Helicobacter pylori* infection among Aborigines (the

Orang Asli) in the northeastern region of Peninsular Malaysia. Am J Trop Med Hyg. 2010;83:1119–22.

30. Pandeya N, Whiteman DC. Prevalence and determinants of *Helicobacter pylori*sero-positivity in the Australian adult community. J Gastroenterol Hepatol. 2011 Mar 28; doi: 10.1111/j.1440-1746.2011.06726.x. [Epub ahead of print]

31. Faleh FZ, Ali S, Aljebreen AM, Alhammad E, Abdo AA. Seroprevalence rates of *Helicobacter pylori* and viral hepatitis A among adolescents in three regions of the Kingdom of Saudi Arabia: is there any correlation? Helicobacter. 2010;15:532–7.

32. Ozen A, Furman A, Berber M, Karatepe HO, Mutlu N, Saricoban HE, et al. The effect of *Helicobacter pylori* and economic status on growth parameters and leptin, ghrelin, and insulin-like growth factor (IGF)-I concentrations in children.Helicobacter. 2011;16:55–65.

33. Thankachan P, Muthayya S, Sierksma A, Eilander A, Thomas T, Duchateau GS, et al. *Helicobacter pylori* infection does not influence the efficacy of iron and vitamin B(12) fortification in marginally nourished Indian children. Eur J Clin Nutr. 2010;64:1101–7.

34. Cherian S, Burgner DP, Cook AG, Sanfilippo FM, Forbes DA. Associations between *Helicobacter pylori* infection, co-morbid infections, gastrointestinal symptoms, and circulating cytokines in African children. Helicobacter. 2010;15:88–97.

35. Abdollahi A, Morteza A, Khalilzadeh O, Zandieh A, Asgarshirazi M. The role of *Helicobacter pylori* infection in gastro-oesophageal reflux in Iranian children. Ann Trop Paediatr. 2011;31:53–7.

36. Tanih NP, Okeleye BI, Ndip LM, Clarke AM, Naidoo N, Mkwetshana N, et al.*Helicobacter pylori* prevalence in dyspeptic patients in the Eastern Cape province - race and disease status. S Afr Med J. 2010;100:734–7.

37. Aje AO, Otegbayo JA, Odaibo GN, Bojuwoye BJ. Comparative study of stool antigen test and serology for *Helicobacter pylori* among Nigerian dyspeptic patients–a pilot study. Niger J Clin Pract. 2010;13:120–4.

38. Hestvik E, Tylleskar T, Kaddu-Mulindwa DH, Ndeezi G, Grahnquist L, Olafsdottir E, et al. *Helicobacter pylori* in apparently healthy children aged 0–12 years in urban Kampala, Uganda: a community-based cross sectional survey. BMC Gastroenterol. 2010;10:62.

39. Dattoli VC, Veiga RV, da Cunha SS, Pontes-de-Carvalho LC, Barreto ML, Alcantara-Neves NM. Seroprevalence and potential risk factors for *Helicobacter pylori* infection in Brazilian children. Helicobacter. 2010;15:273–8.

40. Miranda AC, Machado RS, da Silva EM, Kawakami E. Seroprevalence of *Helicobacter pylori* infection among children of low socioeconomic level in Sao Paulo. Sao Paulo Med J. 2010;128:187–91.

41. Janjetic MA, Goldman CG, Balcarce NE, Rua EC, Gonzalez AB, Fuda JA, et al. Iron, zinc, and copper nutritional status in children infected with *Helicobacter pylori*. J Pediatr Gastroenterol Nutr. 2010;51:85–9.

42. F, Estrella B, Egas J, Naumova EN, Griffiths JK. The effect of *Helicobacter pylori* infection on growth velocity in young children from poor urban communities in Ecuador. Int J Infect Dis. 2010;14:e788–91.

43. Araf LN, Pereira CA, Machado RS, Raguza D, Kawakami E. *Helicobacter pylori*and iron-deficiency anemia in adolescents in Brazil. J Pediatr Gastroenterol Nutr. 2010;51:477–80.

44. Fialho AM, Braga AB, Braga Neto MB, Carneiro JG, Rocha AM, Rodrigues MN, et al. Younger siblings play a major role in *Helicobacter pylori* transmission among children from a low-income community in the Northeast of Brazil. Helicobacter. 2010;15:491–6.

45. Vendt N, Kool P, Teesalu K, Lillemae K, Maaroos HI, Oona M. Iron deficiency and *Helicobacter pylori* infection in children. Acta Paediatr. 2011 Mar 21; doi: 10.1111/j.1651-2227.2011.02281.

46. Katsanos KH, Tatsioni A, Tsakiris V, Christodoulou D, Tsianos EV. *Helicobacter pylori* is a major public health priority in western Balkans: an endoscopy referral center experience. Eur J Intern Med. 2010;21:306–9.

47. Epplein M, Signorello LB, Zheng W, Peek RM, Jr, Michel A, Williams SM, et al. Race, African ancestry, and *Helicobacter pylori* infection in a low-income United States population. Cancer Epidemiol Biomarkers Prev. 2011;20:826–34.

48. Sonnenberg A, Lash RH, Genta RM. A national study of Helicobactor pylori infection in gastric biopsy specimens. Gastroenterology. 2010;139:1894–901. e2; quiz e12

49. McJunkin B, Sissoko M, Levien J, Upchurch J, Ahmed A. Dramatic Decline in Prevalence of *Helicobacter pylori* and Peptic Ulcer Disease in an Endoscopy-referral Population. Am J Med. 2011;124:260–4

50. Olokoba, A. B., O. A. Obateru, and M. O. Bojuwoye. "*Helicobacter Pylori*Eradication Therapy: A Review of Current Trends." *Nigerian Medical Journal Journal of the Nigeria Medical Association* 54.1 (2013): 1–4. *PMC*. Web. 12 Oct. 2017.

51. Cardenas VM, Mena KD, Ortiz M, Karri S, Variyam E, Behravesh CB, et al. Hyperendemic *H. pylori* and tapeworm infections in a U.S.-Mexico border population. Public Health Rep. 2010; 125:441–7.

52. Kirchner GI, Beil W, Bleck JS, Manns MP, Wagner S. Prevalence of *Helicobacter pylori* and occurrence of gastroduodenal lesions in patients with liver cirrhosis. Int J Clin Exp Med. 2011;4:26–31.

53. Senbanjo I, Akinbami A, Diaku-Akinwumi I, Oshikoya K, Adeyemo T, Dada O, et al. *Helicobacter pylori* infection among a pediatric population with sickle cell disease. J Natl Med Assoc. 2010;102:1095–9.

54. Diamantidis MD, Ioannidou-Papagiannaki E, Kountouras J, Mandala E, Tsapournas G, Frida-Michailidou I, et al. High prevalence of *Helicobacter pylori*infection in Greek patients with myelodysplastic syndromes. Acta Haematol. 2010;124:141–9.

PARAMETERS TO BE EVALUATED

Endoscopic features of chronic gastritis:

Erythema, hyperemia, atrophy, and mucosal nodularity according to the criteria of the Sydney grading system.

Histologic features of chronic gastritis:

Increased lymphocytes and plasma cells in the lamina propria according to the criteria of Sydney grading system.

Histologic features of H. pylori:

1. Comma or S shaped bacilli (2-4 μm long and 0.5-1 μm thick).

2. Bacilli adhered on cell surface or free in mucosa layer, with its typical morphology, and forming at least small colonies.

3. Isolated forms of H. pylori like bacterium are considered negative by histology.

Materials and Methods:

Study population:

This consists of patients with complaints of dyspepsia for more than 6 months at General surgery department, Govt.Kilpauk Medical College and Hospital, Chennai.

Method of collection of data (including sampling procedure):

A. Study design: Cross sectional study

B. Place of study: Govt.Kilpauk Medical College and Hospital, Chennai.

C. Study sample size: **87**

N = 4pq/d^2= 4*85*15/ (7.65)^2**= *87***

p: Prevalence of H. pylori in chronic gastritis (85%)

q: 100-p (100-85= 15%)

d: allowable relative error 9% (9*85/100 =7.65 %)

Confidence level – 95%

D. Study period: December 2016 – September 2017

E. Method of sampling: Random sampling.

F. Inclusion criteria:

1. Patients between the ages of 20 – 60 years
2. Symptomatic patients with epigastric pain, postprandial fullness, heartburns, bloating, belching, nausea, vomiting for minimal period of 6 months.
3. Patients given written informed consent

G. Exclusion criteria:

H.pylori regimen before 4 weeks of endoscopy.

Congestive cardiac failure

MI or chest pain within last 12 months

COPD (FEV less than 1.25, home oxygen use)

Coagulopathy (INR greater than 2) or bleeding disorders

Platelet count less than 75000

H. Methodology:

1. **Sample:** patients will be selected on basis of inclusion and exclusion criteria.
2. **Written informed consent** will be taken (Attached below).
3. **PROFORMA:**

    1. PATIENT NAME:

    2. IP No:

    3. Department:

    4. Hospital:

    5. Age:

    6. Sex:

7. Address:

8. Contact No:

9. Occupation:

10. Chief complaints:

11. Past history:

12. General examination:

13. Vitals:

14. Abdominal examination:

15. Cardiovascular and Respiratory system examination:

16. Investigations:

    Complete hemogram

    Renal function tests and Electrolytes

    Liver function test

    BT/CT

    VCTC/HbSAg

    Chest X-ray and Abdomen X-ray

17. Specific investigations:

USG abdomen and pelvis

Echocardiogram

Endoscopic biopsies

5 biopsies taken from following sites:

1. 1 from antrum 2-3 cm from pylorus lesser curvature,
2. 1 from antrum 2-3 cm from the pylorus greater curvature,
3. 1 from the corpus, 8cm from the cardia lesser curvature,
4. 1 from the corpus 8cm from the cardia greater curvature,
5. 1 from the angularis.

Histopathology examination:

Biopsied specimen are sent to histopathology by preserving in 10% buffered formalin solution.

சுயஒப்புதல்படிவம்:

ஆய்வுசெய்யப்படும்தலைப்பு:

நாள்பட்டஇரைப்பைஅழற்சியில்எச். பயிலொரிகிருமியின்நோய்தாக்கம்குறித்தஓர்ஆய்வு

PREVALANCE OF H. PYLORI IN CHRONIC GASTRITIS

ஆய்வுசெய்யபடும்துறை : பொதுஅறுவைசிகிச்சைதுறை

மருத்துவமனை: அரசுகீழ்பாக்கம்மருத்துவகல்லூரி
மருத்துவமனை

பங்குபெறுபவரின்பெயர் :

பங்குபெறுபவரின்வயது:

பங்குபெறுபவரின்மருத்துவமனைஎண்:

பங்குபெறுபவர்இதனை (✓)குறிக்கவும்:

1.
எச்பயிலொரிகிருமியைகண்டறியவாய்வழியாகஉழாய்போட்டுவயிற்றைஆய்வுசெய்துசிறுதிசுவைஎடுத்து பரிசோதனைசெய்துஅதன்பின்புஅதற்கானமுறையானசிகிச்சையைமேற்கொள்ளலாம்என்றுமருத்துவர்கூற அறிந்தேன்.
மேலும்என்னுடையசந்தேகங்களைகேட்கவும்அதற்கானவிளக்கங்களைபெறவும்வாய்ப்பளிக்கப்பட்டது.
( )

2. நான்இந்தஆய்வில்தனிச்சையாகதான்பங்கேற்கிறேன்.
எந்தகாரணத்தினாலோநான்இந்தஆய்வில்இருந்துவிலகஆசைப்பட்டால்எந்தபிரச்சனையும்இன்றிவிலகலாம்என்றும்அறிந்துகொண்டேன். ( )

3.
இந்தஆய்வுசம்பந்தமாகவோஇவைசார்ந்தமேலும்ஆய்வுமேற்கொள்ளும்பொழுதோஇந்தஆய்வில்பங்குபெரும்மருத்துவர்என்னுடையமருத்துவஅறிக்கைகளைபார்ப்பதற்குஎன்அனுமதிதேவையில்லைஎனஅறிந்தேன். ( )

4. இந்தஆய்வில்பங்குகொள்ளநான்சுயநினைவோடும்முழுசம்மதத்தோடும்ஒப்புக்கொள்கிறேன்.( )

பங்குபெறுபவரின் ஆய்வாளரின்பெயர்;:

பெயர்:

பங்குபெறுபவரின் ஆய்வாளரின்கையொப்பம்:

கையொப்பம்:

Name	Age	Sex	OP NO	Endoscopic Finding	Chronic GastritisH	pylori	Smoking	Alcohol	Tobacco	Diet
srinivasan	32	m	4526	gastritis	1	1	1	1	1	1
vi0th	40	m	6953	gastritis	1	1	1	1	1	1
rani	32	f	3625	0rmal	0	0	0	0	0	1
saravanan	35	m	9654	gastritis	1	1	1	1	1	1
periyasamy	55	m	3256	oesophagitis	0	0	0	0	0	1
amritha	37	f	4852	gastritis	1	1	0	0	0	1
rajesh	21	m	1563	gastric ulcer	0	0	0	0	0	1
dinesh	25	m	5489	gastritis	1	0	1	1	1	1
rajkumar	44	m	6659	0rmal	0	0	0	0	0	1
iyappan	38	m	3254	0rmal	0	0	0	0	0	1
shanthi	40	f	2596	gastritis	1	1	0	0	0	1
shenbagavalli	31	f	1254	gastritis	1	1	0	0	0	1
pachaiyammal	41	f	3300	0rmal	0	0	0	0	0	1
kaja moideen	56	m	5962	gastritis	1	1	1	1	1	1
unnamalai	33	m	3620	gastritis	1	1	1	1	1	1
kamatchi	35	f	1520	0rmal	0	0	0	0	0	1
krishnaveni	42	f	1852	gastritis	1	1	0	0	1	0
thanikachalam	48	m	3620	gastritis with gastric ul	1	0	0	0	0	1
rajesh	47	m	9874	gastritis	1	1	1	1	1	1
selvaraj	27	m	6523	gastritis	1	0	1	1	1	1
pushpalatha	43	f	2214	gastritis	1	1	0	0	0	1
vijaya	44	f	5698	gastritis	1	1	0	0	1	1
shalini	47	f	3699	gastritis	1	1	0	0	1	1
kathavarayan	57	m	2588	gastritis	1	0	1	1	1	1
chakrabani	37	m	8523	0rmal	0	0	0	0	0	1
papitha	45	f	8520	gastritis	1	1	0	0	1	1
narayanan	26	m	7410	gastritis	1	0	1	1	1	1
yasar	29	m	1596	oesophagitis	0	0	0	0	0	1
babu	45	m	3524	gastritis	1	1	1	1	1	1
gunasekar	44	m	1856	gastritis	1	0	1	1	1	1

ramesh	22	m	6851	duodenitis	0	0	0	0	0	1
michael	24	m	3206	gastritis	1	0	1	1	1	1
nagappan	34	m	9996	gastritis	1	1	1	1	1	1
ajay	26	m	3326	gastritis	1	1	1	1	0	1
kalyani	36	f	5586	0rmal	0	0	0	0	0	1
paneerselvam	46	m	4448	gastritis	1	1	1	1	1	1
manjunathan	45	m	3693	gastritis	1	0	1	1	1	1
janarthanan	28	m	2583	0rmal	0	0	0	0	0	1
nagaraj	32	m	8888	0rmal	0	0	0	0	0	1
raghavi	46	f	4356	gastritis	1	1	0	0	1	1
sulthana	48	f	6524	gastritis	1	0	0	0	0	1
kamini	27	f	9653	gastric ulcer	0	0	0	0	0	1
mala	49	f	3256	oesophagitis	0	0	0	0	0	1
rajeshwari	44	f	4458	gastritis	1	0	0	0	0	1
mariyammal	54	f	3765	gastritis	1	1	0	0	1	1
govindan	36	m	9994	0rmal	0	0	0	0	0	1
durairaj	47	m	5912	gastritis	1	1	1	1	1	1
malliga	44	f	5000	0rmal	0	0	0	0	0	1
lokeshwari	29	f	6748	gastritis	1	0	0	0	0	1
rathinammal	57	f	6895	gastric ulcer	0	0	0	0	0	1
siraj	37	m	3562	gastritis	1	1	1	1	1	1
kumari	43	f	9563	gastritis	1	1	0	0	1	1
kamlesh	48	m	3201	gastritis	1	1	1	1	1	1
kavi priya	22	f	4859	0rmal	0	0	0	0	0	0
sugandhan	31	m	2630	gastritis	1	1	1	1	0	1
arun	49	m	7529	0rmal	0	0	0	0	0	0
saravanan	30	m	3652	duodenal ulcer	0	1	0	0	0	1
philips	32	m	5963	gastritis	1	1	1	1	0	1
maariyammal	47	f	1115	0rmal	0	0	0	0	0	1
shamsath begum	45	f	2222	gastritis	1	1	0	0	1	1
regina	28	f	3598	gastritis	1	1	0	0	0	1

divakar	35	m	6359	gastritis	1	0	1	1	1	1
muthuraj	47	m	4853	0rmal	0	0	1	0	1	1
kaliyaperumal	59	m	8639	gastric cancer	0	1	0	0	0	1
kumar	37	m	5328	gastric ulcer	0	1	0	0	0	1
vinitha	46	f	2614	0rmal	0	0	0	0	0	0
thanraj	35	m	5362	oesophagitis	0	1	0	0	0	1
valliammal	39	f	2954	0rmal	0	0	0	0	0	1
shobana	26	f	5263	gastritis	1	1	0	0	0	1
girija	33	f	2893	gastritis	1	1	0	0	0	1
khaleel	41	m	4265	duodenal ulcer	0	1	0	0	0	1
vijay	36	m	1220	gastritis	1	0	1	1	1	1
srinivasan	42	m	9523	gastritis	1	1	1	1	1	1
prema	34	f	5863	gastritis	1	1	0	0	1	1
vivek	47	m	2963	0rmal	0	0	1	1	0	1
vishnu	43	m	8593	gastritis	1	1	1	1	1	1
chitra	38	f	1453	gastritis	1	1	0	0	1	1
ragini	24	f	3625	0rmal	0	0	0	0	0	1
varadharajan	44	m	1563	gastritis	1	1	1	1	1	1
aruna	39	f	4896	duodenitis	0	1	0	0	0	1
amreen	37	f	2631	gastritis	1	0	0	0	0	1
jesintha	35	f	2635	gastritis	1	1	0	0	1	1
krishnamoorthy	46	m	4896	gastritis with duodenit	1	1	1	1	1	1
balambigai	33	f	3214	gastritis	1	0	0	0	0	1
parvez	23	m	2368	0rmal	0	0	1	1	0	1
mythili	30	f	8216	gastritis	1	0	0	0	0	1
shenbagam	44	f	1230	gastritis	1	1	0	0	1	1